ABSTRACT

OF

The Proceedings of the Forty-third Annual Meeting of the Association of Life Insurance Medical Directors of America

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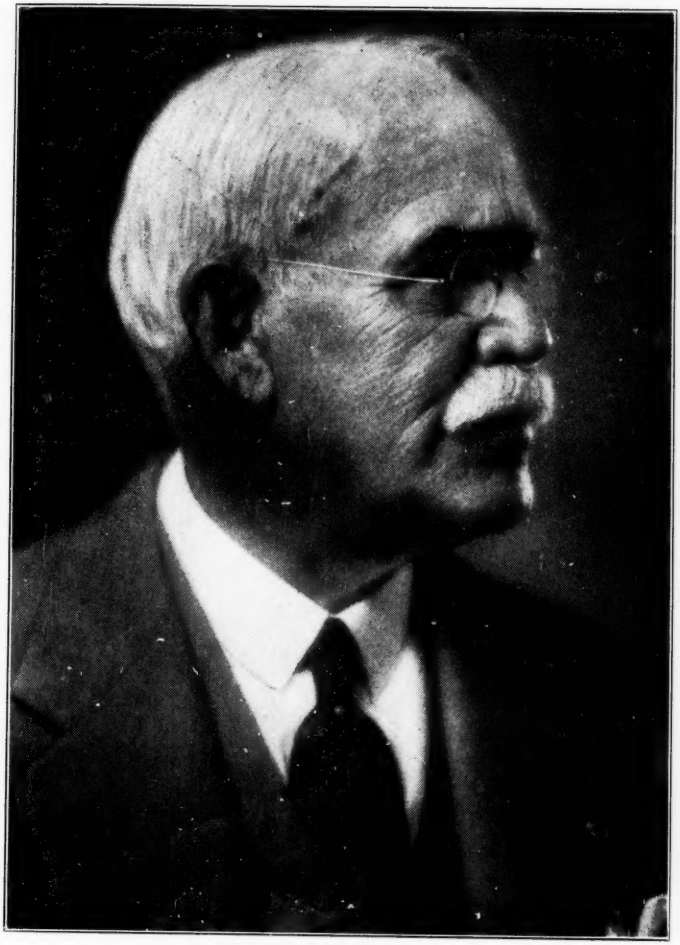

OSCAR H. ROGERS, M. D.
Chairman of M. I. B. Committee 1902-1932

An Abstract of the Proceedings

OF THE

Association of Life Insurance Medical Directors of America

FORTY-THIRD ANNUAL MEETING

The Forty-third Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Waldorf-Astoria Hotel, New York City, on October 27 and 28, 1932.

The following members, delegates and guests were present at some time during the sessions: Drs. J. W. Abbott, C. D. Alton, H. H. Amiral, H. B. Anderson, T. D. Archibald, E. M. Armstrong, T. M. Armstrong, W. B. Aten, A. E. Awde, H. A. Baker, H. A. Bancel, G. H. Barber, W. B. Bartlett, J. T. J. Battle, W. W. Beckett, M. B. Bender, S. R. Benedict, J. R. Biggs, C. C. Birchard, A. B. Bisbee, W. F. Blackford, N. R. Blatherwick, William Bolt, J. T. Bowman, W. M. Bradshaw, F. G. Brathwaite, J. L. Brooks, C. T. Brown, H. B. Brown, B. F. Byrd, J. T. Cabaniss, Saro Cali, E. J. Campbell, F. H. Carber, H. R. Carstens, V. S. Caviness, L. D. Chapin, Louis Chargin, C. L. Christiernin, C. P. Clark, E. A. Colton, H. W. Cook, P. M. Cort, H. C. Cruikshank, George Cullen, R. M. Daley, H. D. Delamere, P. G. Denker, E. J. Dewees, E. G. Dewis, F. F. Dexter, C. R. Dudley, J. H. Dunkley, O. M. Eakins, J. L. Evans, Franklin Everett, J. G. Falconer, H. H. Fellows, W. E. Ferguson, Otto Folin, R. A. Fraser, F. I. Ganot, H. W. Gibbons, A. D. Ghiselin, R. A.

Goodell, F. L. Grosvenor, F. W. Hagney, Llewellyn Hall, Frank Harnden, W. C. Hausheer, W. D. Heaton, C. A. Heiken, W. L. Hilliard, D. W. Hoare, C. E. Homan, J. C. Horan, Ross Huston, W. G. Hutchinson, C. E. Iliff, A. S. Irving, C. B. Irwin, W. A. Jaquith, B. Y. Jaudon, A. O. Jimenis, A. E. Johann, G. E. Kanouse, C. P. Kautzman, A. S. Knight, F. H. Lahey, W. P. Lamb, A. J. Lotka, F. M. McChesney, F. H. McCrudden, C. B. McCulloch, George McLean, W. T. McNaughton, F. W. McSorley, A. A. MacDonald, L. F. MacKenzie, W. L. Mann, W. H. Miller, W. L. Miller, J. T. Montgomery, R. C. Montgomery, E. B. Mountain, William Muhlberg, A. E. Murphy, C. T. Necker, John Nevin, C. F. Nichols, J. B. Nichols, E. C. Noble, W. L. O'Connell, M. I. Olsen, G. W. Parker, J. A. Patton, C. A. Peters, J. S. Phelps, C. B. Piper, J. E. Pollard, W. W. Quinlan, C. R. Rein, P. V. Reinartz, W. A. Reiter, F. P. Righter, A. J. Robinson, T. H. Rockwell, Hugo Roesler, O. H. Rogers, H. B. Rollins, E. K. Root, R. L. Rowley, C. L. Rudasill, S. C. Rumford, E. F. Russell, H. C. Scadding, C. E. Schilling, S. B. Scholz, Jr., B. T. D. Schwarz, G. H. Shaw, J. T. Sheridan, A. L. Sherrill, D. M. Shewbrooks, R. L. Shields, R. R. Simmons, J. L. Siner, DeWitt Smith, Morton Snow, H. F. Starr, David Steel, J. B. Steele, D. F. Steuart, I. R. Stidger, A. C. Stokes, A. R. Stone, S. J. Streight, E. V. Sweet, B. C. Syverson, L. G. Sykes, H. F. Taylor, W. E. Thornton, I. G. Towson, Wallace Troup, H. A. Turner, J. P. Turner, H. E. Ungerleider, Euen Van Kleeck, W. R. Ward, F. S. Weisse, F. L. Wells, P. V. Wells, D. E. W. Wenstrand, C. D. Wheeler, C. F. S. Whitney, T. H. Willard, R. L. Willis, J. A. Wilson, G. E. Woodford, L. S. Ylvisaker, and Messrs. H. R. Bassford, A. J. Blaivas, R. G. Clarke, L. I. Dublin, R. E. Fletcher, Arthur Hunter, Herbert Marks, Wendell Strong, and M. W. Torrey.

Total attendance at all sessions, 188.

During the past year, nine members of our Association have died. The following testimonials to their memory have been prepared by representatives of the Association.

DR. THOMAS W. BICKERTON

Dr. Thomas W. Bickerton, Medical Director of the New York Life Insurance Company, and a member of the Association of Life Insurance Medical Directors of America since June, 1899, died at his home in Fanwood, New Jersey, on May 4, 1932.

He was born at Cleveland, Ohio, on July 12, 1853, and received his early education at the St. Timothy Hall Preparatory School, Catonsville, Maryland. He attended Columbia and was graduated from the College of Physicians and Surgeons of that University in 1881. He married Miss Eva N. Garretson, of New Brunswick, New Jersey. Mrs. Bickerton died in August, 1921. There were no children.

During the early years of his practice of medicine in New York City, Dr. Bickerton was Resident Physician of the Colored Home and Hospital, Consulting Physician of the Lincoln Hospital, member of the Staff of the Eastern Dispensary Lying-in Hospital and of the New York Dispensary, and held other responsible Staff positions in New York City medical institutions. He was a member of the New York County Medical Society, and for several years was Treasurer of the Widows and Orphans Fund of that Society. He was also a member of the American Medical Association.

In January, 1889, he became an examining physician of the New York Life Insurance Company, and a member of its Medical Board in 1895. In January, 1899, he was appointed Assistant Medical Director of the New York Life, and Medical Director of that Company in May, 1912. For over thirty years he had charge of all New York Life medical examiners and examinations in the Metropolitan district in New York City and was also called upon to give opinions on many important cases in the Company's general business throughout the country, and in these exacting duties his judgment, knowledge and experience were highly esteemed. In January, 1931, he retired from active participation in his duties. During his long association with the Home Office Executives, the Medical Department, and the Field Force, he won the affectionate regard and respect of his associates, and enlisted

a remarkable personal attachment and popularity, and his services to the New York Life Insurance Company were most faithfully, efficiently, and meritoriously performed.

DR. HENRY COLT

Dr. Henry Colt, Medical Director of the Berkshire Life Insurance Company, died in his sleep during the night in his home in Pittsfield, Massachusetts, December 8, 1931, in his 76th year. His death was very unexpected because he had been active up to the day before.

Dr. Colt was born in Pittsfield, November 9, 1856, son of Henry and Elizabeth (Bagon) Colt. He prepared for college at the Pittsfield High School. He entered Williams College in 1874 and was graduated in the class of 1878. Upon finishing at Williams, Dr. Colt entered the Harvard Medical School from which he was graduated in 1884. Thereafter he began his practice in the Berkshire Life Building, in which building his office was still located at the time of his death. Dr. Colt never married.

Dr. Colt had been Medical Director of the Berkshire Life Insurance Company since October, 1901, and had served as a Director of the Company from January, 1905. He was also medical examiner for Berkshire County Second District, chairman of the medical board of the House of Mercy Hospital, president of the Pittsfield Anti-tuberculosis Association and of the Berkshire Athenæum and Museum, president of the Berkshire County Red Cross and long of the city's chapter, had served as president of the Medico-Legal Society of Massachusetts, as president of the Berkshire District Medical Society and had membership also in the State Organization. He was also interested in the Berkshire County Home for the care of crippled and deformed children.

Dr. Colt had many activities outside his profession. For 16 years he was president of the Country Club of Pittsfield, a leader in Rotary, director of the Pittsfield Third National Bank and Trust Company, a member of the First Congregational Church and also active in the Men's Club of that church.

Tribute from the District Attorney, Thomas F. Moriarty of Springfield, well describes Dr. Colt:

"Dr. Henry Colt faithfully and efficiently served Berkshire County as medical examiner for many years. He had a fine and well-deserved reputation as a man of integrity and a doctor of ability. His duties brought him in close touch with the District Attorney's Office and his work was effective and of great aid to me and my office. Berkshire has lost a distinguished son and a faithful public official."

DR. JOSEPH BRAINARD HALL

It is with the deepest regret that I bring to your attention the death, on May 24, 1932, of Dr. Joseph Brainard Hall, Medical Director of The Connecticut Mutual Life Insurance Company of Hartford, and a member of this Association.

Dr. Hall was born in Hartford in 1868, the son of Joseph and Orvilla (Basset) Hall. His parents came to Hartford from Ashfield, Massachusetts, when his father became a teacher at the Hartford Public High School of which he later became the principal. Dr. Hall attended the Hartford Public Schools and in 1887 graduated from the Hartford Public High School. Following this he went to Sheffield Scientific School of Yale University from which he graduated. Following his graduation he attended the Yale College of Medicine from which he received his medical degree in 1892. From 1892 to 1894 he interned at the Hartford Hospital. Later he became Assistant Visiting Physician to the same institution.

In his undergraduate days at Yale Dr. Hall was active in athletics, a member of the boxing squad and of the baseball varsity and prominent in tennis, in which sport he was keenly interested and well-known even after his college days.

In 1907, Dr. Hall was appointed Medical Director of The Connecticut Mutual Life Insurance Company, which office he held till his death. Prior to this he had acted as Assistant Medical Director for a period of several years. His earliest association with life insurance work was with the old Hartford

Life. He was a member of the Descendents of the Mayflower Association, St. Johns Lodge A. F. and A. M., The Hartford Club, The Hartford Golf Club and the Hartford Medical Society.

Dr. Hall leaves his wife, Mrs. Elizabeth Wainwright Hall, and an only son, Joseph Talcott Hall. Those of us who have had the privilege of knowing Dr. Hall all feel a deep and personal loss in his passing from our midst.

DR. FRANCIS A. McGREEN

With sincere regret we report that Dr. Francis A. McGreen, Medical Supervisor of the New York Life Insurance Company, died at the Memorial Hospital in New York City on December 28, 1931.

Dr. McGreen was born at Bellows Falls, Vermont, September 17, 1884. He attended Brown University, Providence, R. I., and then the Yale Medical School, where he was Editor of the Yale Medical Journal. He then entered the College of Physicians and Surgeons of Columbia University and there received his medical degree in 1914. He served for several years in various hospitals and practiced medicine in New York City and vicinity and then entered the service of the New York Life Insurance Company as a medical examiner and member of the Medical Board. He was appointed Medical Supervisor in 1924 and was elected a member of the Medical Directors' Association in 1929. He was a member of the New York State and New York County Medical Associations.

Dr. McGreen, from the first, showed serious interest in and complete comprehension of insurance medicine and applied very successfully his thorough medical experience to the selection and valuation of life insurance applicants and the impairments encountered in life insurance selection. He was always held in the greatest esteem and friendship by his associates in the New York Life Insurance Company, who recognized the sterling qualities of his character, his integrity and continual willingness to help his associates.

DR. THOMAS F. McMAHON

Dr. Thomas F. McMahon, for many years Medical Referee of the Manufacturers Life Insurance Company, died on October 25, 1931. Dr. McMahon graduated from the University of Toronto and after post graduate work in Dublin settled in Toronto, where he built up a very large practice. He was examiner for the Manufacturers Life Insurance Company and in June, 1908, was appointed Assistant Medical Referee, becoming Medical Referee of the Company in 1911. He was also Chief Examiner in Toronto for the Mutual Life Assurance Company of Canada and Medical Referee of the Equity Life.

In his later years he devoted his time almost entirely to insurance work and as a result became one of the leading insurance medical authorities in Canada whose opinion both as examiner and referee was valued by the Medical Directors of all the Canadian Companies. He was long a member of this association and was the first Canadian to be honored with the presidency. The programs which he arranged for the meetings over which he presided, bear witness to the keenness of his interest in his chosen work.

He retired from the service of the Manufacturers Life in 1927 and the last few years of his life unfortunately were marred by ill-health, although his cheery manner and native wit allowed him to hide his illness from any but those who knew him best.

He was a churchman of unswerving faith and the amount of medical work which he did among the poor of the city was large. His standing in the medical profession was attested by the fact that he was honored with life membership in the Academy of Medicine, Toronto, an honor reserved only for those who have served their profession well. He was a popular figure not only at Agency Conventions but at social gatherings of any kind. His interests were wide and varied and he was respected and beloved alike by patients, friends and fellow practitioners.

DR. ARCHIBALD MERCER

Dr. Archibald Mercer, who died on November 3, 1931, was a life long resident of Newark, New Jersey. In fact, he died in the same room in which he was born eighty-three years ago. His father, Dr William Theodore Mercer, was also a Newark physician and was related to Dr Hugh Mercer of Revolutionary fame. His mother, Gertrude Ann Frelinghuysen, was a direct descendant of General Frederick Frelinghuysen, also of Revolutionary fame, and a sister of Frederick T. Frelinghuysen, who was Secretary of State during the administration of President Arthur.

Dr. Mercer received his academic education in the Newark Academy and Rutgers College and his medical education in the College of Physicians and Surgeons of New York from which institution he was graduated in 1871. His connection with the Newark hospitals and with other Newark institutions was large and varied. Upon his graduation he served as house physician at St. Barnabas Hospital, later becoming Medical Superintendent. He also served as United States examining surgeon for pensions and as police surgeon for the City of Newark.

Dr. Mercer rendered a continuous service in medical societies from the time of his graduation until shortly before his death. For twenty-six years he was the secretary of the Essex County Medical Society and subsequently was elected its president. For thirty years he was the treasurer of the New Jersey State Medical Society and was also elected the president of that organization.

In public life Dr. Mercer rendered service as secretary and later as president of the Society for the Relief of Widows and Orphans of Medical Men of New Jersey, and as a trustee of the Free Public Library and the Newark Museum He was also a member of the Sons of the American Revolution and a life member of the New Jersey Historical Society. Dr. Mercer was one of the oldest communicants of Trinity Cathedral of Newark, to which church his family has been attached since 1792.

In 1904, Dr. Mercer entered the Home Office service of the Mutual Benefit Life Insurance Company and was subsequently appointed an Assistant Medical Director, which position he held at the time of his death.

DR. CHARLES AIKEN VANDERVOORT

Dr. C. A. VanDervoort, Medical Director of the The Fidelity Mutual Life Insurance Company, died July 5, 1932, at the Bryn Mawr Hospital, Philadelphia. While his last illness was of comparatively short duration, Dr. VanDervoort had been in failing health for a long time and had been unable to attend to his office duties for over a year.

Born in Stirling, Ontario, March 11, 1867, Dr. VanDervoort came to Philadelphia as a young man for treatment for a minor injury to his back. His experience in the hospital gave purpose to his ambition to be a doctor, with the result that he graduated in medicine from the University of Pennsylvania with the class of 1893.

Following graduation he served as resident physician in Blockley Hospital, Philadelphia, and then did postgraduate work in ear, nose, and throat in Glasgow. Afterward he was connected with the Episcopal and Polyclinic Hospital in Philadelphia and for some time was surgeon in ear, nose, and throat at St. Christopher's Hospital.

During this period he became medical examiner for several companies, including the Fidelity, and July 1, 1915, became associated with the Head Office Medical Department. On April 15, 1920, he became Assistant Medical Director and in January, 1931, Medical Director.

Dr. VanDervoort had two hobbies which occupied his outside interests—golf and music. He was a member of the Alpha Mu Pi Omega fraternity, the Philadelphia County Medical Society, the American Medical Association, the Philadelphia Country Club, the Canadian Society, and a Director of the North Philadelphia Trust Company.

Dr. VanDervoort was a genial, kindly man, highly regarded in his profession and esteemed as a friend by all his associates. His memory will always be held in affectionate remembrance by his friends and co-workers.

DR. WILLIAM H. E. WEHNER

Dr. William H. E. Wehner, for nearly seventeen years Medical Director of the Fidelity Mutual Life Insurance Company, and a member of this Association, died at the Germantown Hospital, Philadelphia, December 5, 1931.

Dr. Wehner was born in Germantown, March 17, 1865, the son of a prominent Germantown physician. After attending the Philadelphia schools, he entered Jefferson Medical College and was graduated in 1887. Following graduation he served as resident physician at the Philadelphia General Hospital. After practicing in Germantown for several years Dr. Wehner began his career of life insurance medicine as a member of the Medical Department of the Penn Mutual Life Insurance Company, where he remained until 1915, when he was made Medical Director of the Fidelity Mutual. His wise and conservative handling of the selection problem was of real value to the company.

Dr. Wehner was widely known and beloved in the medical circles of Philadelphia. He was a member of the County Medical Society, Medical Examiners' Association, and American Medical Association. He was a Mason and a member of the University Lodge, which is noted for its roster of famous men in the life of Philadelphia.

Dr. Wehner will be remembered as a genial and kindly figure at our meetings. A true representative of the fine old school of medicine, his quiet manner and perfect courtesy will linger long in the minds of those who were privileged to know him well and intimately.

DR. GORDON WILSON

On Wednesday, October 26, 1932, Dr. Gordon Wilson died suddenly and unexpectedly. He had been in his office for the

usual hours, complained of feeling tired, and had gone to a back room to rest. There arose the necessity to disturb him, but when approached he was unconscious. There thus passed, peacefully and quietly, the spirit of a gentleman.

Born on November 30, 1876, a son of the late John A. Wilson and Ellen Gordon Wilson, he was related to many prominent Virginia and Maryland families.

As a child he spent some time at school in Lausanne, and later graduated from the Episcopal High School of Virginia. He then passed two years in the Academic Department of the University of Virginia, and received his medical degree from the same institution in 1899. He returned to Baltimore, spent the summer as a resident at Mt. Wilson, a hospital devoted to the treatment of infants. After this he worked in the Medical Dispensary of the Johns Hopkins Hospital, for a short time, but later in the same year, 1900, was appointed Resident Physician. He had charge of Dr. Osler's private patients, and thus early came under his influence; this gave direction to his future life. The next year was occupied as a Resident Fellow in Pathology under Dr. Welch, and this was followed by some post-graduate work in Pathology and Medicine.

In 1902 he began to practice, and in the same year began his long association with the Medical School of the University of Maryland. His first appointment was Chief of Clinic, in the Medical Dispensary. Each succeeding year, with his enlarging clinical experience, and his developing ability as a teacher, saw continued advancement, until 1913, when he was made Professor of Medicine, and Head of the Medical Department. These onerous responsibilities, including as they did didactic lectures, clinics and administrative direction of medical teaching in the school and hospital, were continued until 1924. From then until now, he occupied a minor but important part in teaching, principally in clinics to the senior classes.

His force and attractiveness as a teacher will ever be remembered by his many students. Thorough in examination, quick in diagnostic perception, clear in exposition, with a deep toned and pleasant voice, he was in many respects the embodiment of

the ideal teacher. He had a retentive memory and was able to use the results of his wide experience, both in reading and practice.

In 1906, he spent four months in Strassburg, working both in pathology and medicine, under the late Professors Chiari and Krehl. Upon returning he had an added enthusiasm for the study of the clinical side of tuberculosis. This was perhaps largely due to two factors, first, his personal experience with the disease; and secondly, the intense interest in that subject in the early 1900's, especially through an exhibition in Baltimore and the meeting of the International Congress in Washington in 1908. In 1909, he was appointed Visiting Chief of the Baltimore Municipal Hospital for Tuberculosis. He, also, was a member of the Board of Managers of the Maryland State Tuberculosis Sanatorium when it first opened in 1910, and this relationship has continued to the present. The mention of these activities serves to indicate his rather important position in relation to this subject in the City and State.

This interest, too, led to his election to membership in the American Climatological and Clinical Association. In 1924, he served as its president, and since then has played an active part in the affairs of this society.

This long interest in the subject of tuberculosis gave color to his entire medical life. Indeed, at one time, he very seriously considered devoting himself entirely to this work. It afforded opportunity for an ever widening group of professional friendships. It provided outlet for an innate sympathy and understanding in his contacts with patients so afflicted. It enabled him, too, with his combination of rare tact and practical advice, to instill courage and hope in many young medical men, victims of tuberculosis in the early part of their professional lives. Not a few such men, now in responsible positions in the tuberculosis world, affirm their love and gratitude for his inspiring guidance, and his help in "placing" them where they have been able to work out their own salvations.

Another important activity, carried out hand in hand with these, was his position as Medical Director of the Maryland

Life Insurance Company of Baltimore. This work had engaged him since 1912. It was only "part time", but it required a study of the problems associated with medical selection in Life Insurance. He was a welcomed and active participant in the annual meetings of the Medical Directors Association, from time to time reading papers particularly relating to the clinical side of this field. Here, too, he enjoyed a wide group of professional friendships, apart from those acquired in the circles of teaching, tuberculosis and the climatological society.

Another phase of his life was his experience during the War Period. In the early part, in 1917, he was assigned to special Tuberculosis Boards, first, for the Maryland Infantry, and later at Gettysburg in a wider field. In the Autumn of the same year, he was sent to the Base Hospital at Camp Meade, Maryland. He had the rank of Captain. This winter was one of extreme severity, quarters were crude and incomplete, living conditions made physical comfort impossible Partially as a result of this, but chiefly as a result of a lack of robustness, which should have prevented his induction into service, his health declined to such degree, that certain of his friends became so apprehensive that they secured orders for a physical survey, and he was discharged from the army. This was all done without his knowledge or consent. When discharged he had the rank of Major.

After this he gradually returned to work and to a reasonable degree of health. Much of his active hospital work was discontinued, especially in tuberculosis. His interest was maintained by his continuance upon the Board of the State Sanatorium. He had an active office and consulting practice which rapidly extended, especially since so much of his time was no longer occupied in teaching. Recently, he spent one day in every other week as a Medical Consultant to the Veterans Bureau Hospital at Mount Alto, Washington. This work he greatly enjoyed.

In 1908, he married Miss Elizabeth Preston Elliott, daughter of the late Mr. and Mrs. Warren G. Elliott of Baltimore. Mrs. Wilson survives, together with two children, Mrs. John Oliver Needles and Miss Elizabeth Elliott Wilson. During most of

his married life he lived at 12 Whitfield Road, Guilford, Baltimore, in a house largely designed by himself, and which always remained one of his minor enthusiasms.

He was a man of many qualities, but perhaps his chief characteristic was his abundant success in friendships. This was, indeed, a rare gift, arising from a deep sympathy and quick appreciation, coupled with a gracious and courteous manner. Yet, it was of no facile type as he was fearless in judgment and quick in resenting any deviation of right conduct. He gave much but, in the same spirit, demanded much. He was singularly clean in thought and expression; with a keen wit and an appreciative humor. His hearty laugh and resonant voice will be sadly missed.

Life for him was relatively short, but, as it was lived, full. From all its facets, husband, father, friend, counsellor, teacher, colleague, dinner companion, crowd memories of his endearing charm. At its ending, his "luck" held good. He had wished for a sudden death; this was granted.

Often did he quote snatches or tags of pœtry, perhaps most frequently from his longtime favorite, Kipling. And from Kipling we may get a most appropriate estimate:

"And as he trod that day to God, so walked he from his birth,
In simpleness, in gentleness, in honor, and clean mirth."

Dr. Christiernin—Before proceeding with our program, I wish to extend a most hearty and cordial welcome to our medical friends of the Associate Companies who accepted the invitation to attend the scientific meetings of the Association. I hope that they will feel at home with us, and will partake freely in the discussion of the papers.

This brings us to the scientific portion of our program. The first paper will be presented by Dr. Fellows on "Procedures for Handling of X-ray and Electrocardiographic Examinations."

PROCEDURES FOR HANDLING X-RAY AND ELECTROCARDIOGRAPHIC EXAMINATIONS

By Haynes Harold Fellows, M. D.

Assistant Medical Director, Metropolitan Life Insurance Co.

The importance of the special examinations of applicants for large amounts of insurance was emphasized by the investigation and report of the Jumbo Risk Committee. Following this report, recommendations were made by the Committee for more elaborate examinations of applicants for large amounts of insurance. These recommendations were accepted by the companies and plans were made to put them into operation in January, 1932. Anticipating the new procedure, a considerable portion of the program of the last meeting of the Association was devoted to the elaboration and discussion of the various plans proposed for more careful study of large risks. Particular emphasis was placed on the study of the circulatory system. The Committee appointed to select qualified examiners to make these special examinations in various cities throughout the country prepared and presented a list of approved examiners. Indeed, everything that could be done to facilitate the new examinations was done in advance of January 1, 1932. In spite of all efforts to make the operation of the new methods of examination simple and acceptable, situations developed that had not been anticipated. It was soon evident that certain standards were needed not only for the estimation of the significance of X-ray and electrocardiographic findings, but also for the technique of their preparation. Even the apparatus used in making the records needed standardization.

With these facts in mind, the President of the Association invited some of the members who had actively participated in the discussion at Hartford to meet and prepare recommendations as to machines, technique and underwriting procedure on the more fundamental conditions as indicated by tracings and films. This

group, consisting of Doctors Birchard, Ungerleider, Taylor, Nichols, Turner and Fellows, met in New York in April of this year, and, after thoroughly discussing the problems involved, reached certain conclusions. At a meeting held a few days ago, this report was elaborated and will be described in detail later in this paper.

We have seen that with increasing frequency the various companies are demanding X-rays and electrocardiograms upon certain applicants for insurance. Some of the home offices are equipped to make these examinations; others are delegating this work to physicians in private practice, but all of us are receiving tracings and films from special examiners not connected with life insurance companies. Some of the interpretations of the X-ray films or electrocardiographic tracings are being made by doctors in the insurance companies, and other companies accept the interpretations of the men who make the examinations. In any event, many films or tracings are encountered which cannot be handled in a dogmatic fashion and classified as strictly positive or strictly negative from a medical or life insurance standpoint. There is a large group of cases which demand a carefully considered specialized medical, clinical and life insurance opinion. As with everything else under the sun, when we deal with opinions, we run into difficulties.

We hope to avoid situations which have arisen in the past where a rendered decision would have been materially different, if the evidence at hand had been correctly understood.

There was one outstanding example recently which may be used to illustrate how things go wrong. A man applied for a large amount of insurance and there were nearly twenty companies involved. This necessitated, in addition to the usual physical examination, an X-ray examination of the chest and an electrocardiographic tracing. In this particular case there were two problems. First, the X-ray was not entirely satisfactory from a technical standpoint, and the first doctor who interpreted the X-ray questioned the significance of a small area in one lung field and consulted with an able chest specialist who gave his opinion of the area involved. There came, thus, into the insurance papers, a note of un-

certainty regarding this particular area. It was thought, possibly, to be tuberculous in character. This adverse opinion was accepted by some companies who did not see the original film and who rejected the case outright, without having seen the evidence. Many of the companies withheld action until they could view the original film and form their own opinion. It developed subsequently that the lungs were considered to be entirely normal and the question of tuberculosis was removed.

The second problem in the case was more difficult. Photostats were made of the electrocardiographic tracing and were interpreted as satisfactory by all but one company from which there was reported an adverse interpretation. Details of this interpretation were contrary to fact and, unquestionably, there was some error involved. It is possible that it was a clerical error; it is possible that the leads of the electrocardiogram were confused, but, at any rate, the rejection was made, the adverse finding reported as an established fact.

This is the type of confusion which we seek to avoid. In cases for large amounts, and where many companies are involved, it would avert a good deal of confusion and save time to have more than one X-ray film of the chest taken, so that an original could be viewed by interested companies. As far as the electrocardiographic tracings are concerned, they should be arranged so that there is no doubt as to the identity of the applicant or as to the sequence of the leads. We have all received in our own offices electrocardiographic tracings with the leads reversed, though correctly numbered, or they have been placed in the holders upside down. One who is familiar with tracings can almost always sort them out and arrange them properly, while one who is at all unfamiliar with the work might be sorely puzzled and could make a costly error. Electrocardiographic tracings and X-ray films should be reviewed only by those competent to review them.

Another problem is to secure, either in our own home offices or from men making special examinations in the field, "readable" X-rays and satisfactory electrocardiographic tracings. We have tried to make clear how we wish these examinations made and, assuming satisfactory equipment and technical ability, the work should im-

prove. We should insist on a high standard of work. The instrument makers should realize that their machines must deliver satisfactory results under given conditions. This, unfortunately, is not a fact at the moment. There is a general demand on the part of physicians throughout the country that the various instrument makers put on the market machines which will give uniform results under uniform conditions. It is very difficult to obtain comparable data now, and we believe that we have the opportunity of exerting some pressure to secure standardized instruments.

We will also be able to select physicians to make these special examinations throughout the country who do work which is satisfactory. There is a good deal of poor laboratory work being done, both because of the lack of experience by those who are making these examinations and because of unsatisfactory instruments. We cannot, as a group, afford to be associated with any individual who is not thoroughly experienced and perfectly competent to make these examinations. We can secure the services of the best physicians to help us in this work and it is our belief that we should demand a high type of work and maintain it.

We all realize that it is necessary to have a routine method of handling these special examinations, both as regards the technique involved in the production of satisfactory evidence, and as regards the interpretation. A beginning has been made and the following report represents the first step.

I. Electrocardiographic Technique

In taking any electrocardiographic tracing for life insurance work, the following conditions should be observed as closely as possible.

1. Film speed should be between 25 and 40 mm. per second with an optimum speed of 30 to 35.

2. Each lead should show the standardization (1 cm. deflection per millivolt). For a satisfactory tracing there should not be more than 1 mm. of overshooting during standardizing, and the instrument should come to rest within 0.02 of a second. Time lines should be present on the tracing. At least eight inches of each lead, including the standardization should be submitted for examination. Leads should be properly numbered.

3. Skin resistance to be satisfactory should be within 1500 ohms, and lower if possible. To secure a low resistance, skin should be scrubbed thoroughly with salt solution, or soap and water and alcohol, if necessary. The electrodes should be moist and hot and

a super-saturated saline solution should be used. The subject should be made entirely comfortable to assure relaxation and should be in a sitting erect or semi-erect position when taking the tracing.

4. The name of the instrument should be noted.

5. The signature of the applicant should be attached for identification.

II. X-ray Technique.

X-ray examination of the chest should be made at a distance of at least 6 feet. Film should be non-inflammable, readable, and should bear the name of the applicant together with the date and the right or left side so marked. Care should be taken to center the applicant, so that the entire thorax will appear on a single film. The name of the instrument should be stated.

III. Photostats of Electrocardiograms

All photostats sent from one Company to another should be—

1. Actual size of tracing

2. Distinct and clear with time lines visible.

IV. Electrocardiograms seem to fall naturally into three general classes.

1. Normal—Those which show no evidence of abnormal cardiac changes.

2. Unfavorable—Those which show changes which warrant declining the risk, even in the presence of a normal history and physical examination. The conditions which come under this heading are:
   a. Auricular fibrillation.
   b. Auricular flutter.
   c. Ventricular tachycardia.
   d. A-V block.
      1. P-R interval (or P-Q if Q is present) 0.22 sec. or more Longest P-R interval considered characteristic.
      2. More advanced degrees of block.
   e. QRS interval 0.11 sec. or more.
   f. QRS notching and/or slurring in all leads in two-thirds of complex farthest from iso-electric line.
   g. QRS low voltage, height less than 5 mm. in lead of greatest amplitude.
   h. T wave low voltage, height 1 mm. or less in lead exhibiting the tallest T waves.
   i. T wave inversion in Lead 1 and/or 2.
   j. S-T deviation 2 mm. or more in all or any leads.

3. Questionable—Findings which can be classed as neither normal nor as strictly pathological in the sense of Group 2.
   a. Variability in successive tracings.
   b. Premature contractions.
   c. A-V conduction (0.20-0.219 sec.).
   d. S.A. block, A-V nodal rhythm, Ventricular escape.
   e. Auricular or nodal tachycardia.
   f. P wave definitely abnormal.
   g. QRS over 20 mm. in height.

h. Abnormal QRS axis deviation—right or left.
i. Large Q-3; more than 25% of the greatest QRS deflection in any lead, right axial deviation must be absent, and showing the following characteristics:
   1. First deflection shall be downward followed by a single positive deflection and nothing further.
   2. More than 50% of the complexes should meet this requirement.
j. T wave changes definitely abnormal (other than rejection h and i).
k. Unusual S-T segment (other than rejection j).

V. Anterior-posterior X-ray examination of the thorax should show the following:

1. Lungs to be normal must be free of gross pathology.
2. Bony thorax should show no gross pathology.
3. Aorta—7 cm. shall be considered the upper limit of normal in size.
4. The transverse diameter of the heart shall not be greater than half the transverse diameter of the thorax to be considered normal. Heart measurements conforming to the above shall be in persons of average or normal stature.

One of the most difficult words to select in the report was that describing an X-ray film as "readable". A "readable" film is a satisfactory film; which means that the physician interpreting the X-ray is able to satisfy himself as to whether it is positive or negative, and, if it is positive, what the possible diagnoses are. Viewing such a film leaves no question in the reviewer's mind in the vast majority of cases as to whether pathology is or is not present.

If the above suggestions are followed, there should be available satisfactory X-ray films and electrocardiographic tracings. The co-operation of the various companies in answering questions which arise from time to time will, we believe, result in much higher type of work in a relatively short time.

In many instances there is very little difficulty in deciding upon those cases in which absolute rejection can be decided from the X-ray and electrocardiographic evidence alone. In these cases there is unquestioned abnormality. But there are many borderline cases. For instance, a middle aged applicant, with average or just over average weight, systolic and diastolic blood pressures at the upper limit of normal, has, upon an X-ray examination of the chest, an aorta and heart at the upper limit of normal, and an electrocardio-

graphic tracing which is suggestive of pathology, but not very definite. This type of case should be appraised upon the entire picture and in this way only can a fair decision be made as to what type of insurance, if any, can be issued.

A more difficult type of case, however, is one in which the entire physical examination and history are negative, and either the X-ray or electrocardiographic tracing gives some evidence of abnormality not found in any other way. If the findings are merely suggestive of definite or serious pathology, we may handle the case in either one of two ways: The case may be issued considering only the results of the history and physical examination, as has been done for years, ignoring the X-ray or electrocardiographic evidence, since it is not definite; or, we may wish to protect ourselves very cautiously and this type of case may be rated or rejected. It seems more reasonable, however, that when the X-ray evidence or electrocardiographic evidence is merely suggestive, that the case be judged upon the older standards and a decision be reached on the basis of history and physical examination with which we have had a considerable experience. One other possibility is offered and that is for each company to have available an able cardiologist or radiologist to whom it can go with the evidence in hand and ask for an experienced opinion which can be added to the insurance angle of the case.

Probably the most important phase of this work upon special examinations is the follow-up of those cases upon which an adverse or questionable decision is rendered because of the evidence presented either by X-ray or electrocardiogram alone. If we look at an X-ray film and see in it definite evidence of pulmonary tuberculosis and the case is otherwise clean, the applicant probably will be rejected. If we do not know how long this applicant lives, we will not know how significant, from the standpoint of mortality, the pathology revealed by the X-ray really is. We have all seen large areas of tuberculosis found accidentally in men and women at a ripe old age, who, as far as they can remember, have never been sick. Also, the electrocardiogram gives us many evidences of definite abnormality. Most of the statistics and conclusions of clinical cardiologists, however, are based on follow-up studies of patients

and we may properly ask the question: Does the electrocardiographic evidence of abnormality always have the same meaning, the same prognosis? We may speculate upon the answers to these questions as we choose, but it is obvious that we don't know. Consequently, our most fruitful source of information in connection with this special type of examination will be in the careful collection of data regarding questionable or rejected cases. There can be no doubt that it is absolutely essential that we start the machinery by which we may be able to secure data upon the significance of findings which are being reported by X-ray and electrocardiographic tracings. This work should be begun immediately. We have done it at the Metropolitan Life Insurance Company for over two and a half years in the following way:

1. If insurance is issued on the basis of the examination, we use the renewal records on the anniversary of the examination to ascertain if the insurance is in force.
2. If the application is declined or if the policy is not placed or later lapses, we ascertain from the papers whether the applicant reports previous insurance in force in any department of the Company, whether Ordinary or Industrial Life Insurance or Accident and Health Insurance. If there is such insurance in force, at the time of application, we ask the department keeping the renewal records whether the insurance is in force at the anniversary of the examination.
3. If there is insurance reported in force in other companies, we inquire on the anniversary of examination through the Record Division if this insurance is still in force.
4. If the insurance previously reported is not in force, we refer the cases to the Ordinary Record Division to find if there has been any subsequent action by this or other companies. If any insurance has been issued subsequently by this or other companies, we trace in the manner already described.

5. A form letter is written to the applicant's physician if his name is given in the application.
6. In cases where we do not have the physician's name or we get no information from the physician, a form letter is sent to the applicant. In both letters assurance is given that the information is to be used for scientific research only, that it will be treated confidentially, that it will not prejudice any subsequent application for insurance.
7. In the few remaining cases in which the methods described above fail to elicit information, we refer the case to a credit agency for direct tracing.
8. When we receive a report of the death of any of these applicants, we secure a copy of the death certificate if it is not available in the claim papers.

This procedure, devised and carried out by the Statistical Division of the Metropolitan Life Insurance Company, is very efficient. Between January 1, 1930, and July 1, 1932, there were 1,100 applicants examined at the Home Office upon whom we carried out special examinations. Of them, 1,050 have been traced and are known to be living; 6 have died; 41 are being traced; and there are only 3 cases which they have been unable to trace; 323 cases have been followed for two years.

A follow-up study of this kind will pay handsomely in a few years. We will have available knowledge which is lacking entirely at the present, as to the significance of findings made possible by the special examinations which we are all having made.

DR. FELLOWS—In the first place, this is not my paper. All I did was act as recording secretary for a group of men who know a great deal more about the details of this business than I do. Last Spring a group of men from this organization got together informally in New York, recognized the various problems that confronted the handling of special examinations and worked out all of the material that you have here in this galley

proof and which will subsequently be printed in an improved form in the proceedings So when I say that it is not my paper, it merely means that all I did was record what had been agreed upon unanimously as a beginning, a point of departure in the handling of a new type of material. The entire history of life insurance medicine or medical science or any new work of today is an aim toward accuracy and decision. That is actually what we are doing when we are asking for these so-called special examinations.

You could talk all day or you could write a book and you would get nowhere beyond about three points of consideration. The first is, On whom do we want special examinations? Second, Where shall we get them and how? Third, What will we do with them after we get them? Now, that is covered in this report.

How we shall get them is at the moment determined by the Jumbo Risk Committee. They have asked for these reports on applicants for large amounts. I am not a good prophet and instead of making a prophecy, I would like to make a bet that as we increase our knowledge and our experience in handling these special examinations, the need for more accurate detailed information will be on much smaller amounts of applicants for insurance applied for than at the moment rather than abolishing or raising the amount. In other words, the tendency will unquestionably be toward getting all the information you can on everybody who applies for insurance. There have been most interesting experiences in the past year or in the past few years on the part of those of us who have been doing this work.

In the second place, on how to get these examinations, is being handled most effectively by Dr. Patton and his committee. There has been a surprising reaction toward the approach of the most eminent specialists in the country when they were asked to do this type of work for us. As far as I know, there have been very few, if any, who have felt that they could be high hat about this and who have not offered to cooperate 100 per cent. plus.

There are a few points that I would like to mention. They are not here but will be in the proceedings. The first is that there has been in some instances a feeling that, as in times past, when an X-ray picture was taken, the man or hospital taking that X-ray picture owned the negative. That is true. It has been passed on by the Courts and the negative is the property of the man or the hospital who makes the picture. However, we shall in the future specify very clearly that those original negatives are the property of the company which asks them, and may I ask here that any of that original material which comes in to your Home Offices be filed and held for future reference. That is being done now. It is the wish of the group that are playing with this problem at the moment that that material be on hand for analysis at times goes on. We will have to learn by experience who is capable and who is not capable of doing the type of work that we want. The committee, I know, is swamped with applications, some by good men and some have been accepted. However, I have sent back X-rays to the doctor who took them and asked him to re-take them. It is a rather embarrassing thing to do, to approach a brother physician and ask him to repeat his work because you just can't interpret it. You have got to do it. I was asked yesterday to interpret an X-ray picture of the pelvis. I swear I didn't know how he took the picture. He must have stood on his head. To make a diagnosis based on that particular X-ray picture just couldn't be done. We must insist on the films or the electrocardiographs or the laboratory material that we get being useful. If it is not useful and the doctor has contracted to give us what we want, we have got to go back at him until we get it, and if the complaints of the individual companies are brought to the attention of Dr. Patton's committee, we will very shortly have a list of perfectly competent specialists who are doing the type of work we want done.

Not all of the fault lies with the men themselves. I don't suppose there is one of us that has not been besieged by manufacturers, and the criteria for the successful taking of an X-ray or electrocardiograph from the standpoint of the manufacturer

seems to be the amount of nickel plate they can put on an instrument and the amount of money they can extract from the buyer. There is no doubt but that this group, working as a unit, will be able to exert enough pressure on instrument manufacturers to get what they want, and, I believe—and I have had some experience which leads me to this belief—at a price which is much more reasonable and equitable than today.

There have been laid down very careful rules for technique, both regarding X-ray and electrocardiographic work. These are going to be available to the special examiners and they will have to be educated to some extent to give us what we want when we ask for it.

I am not going to burden you with the technical end of this thing at all. It is, after all, the purpose of this paper to put in a book readily available the conclusions or the suggestions of those who are doing this work. The technique of taking proper X-ray or electrocardiograph is a standard thing. It is not a by-guess-or-by-gosh thing. It is not made like mother made her biscuits. You have to follow rules of procedure. The rules for a standard instrument can be standardized and our results should be comparable.

The next and probably the most difficult portion of the whole problem, after we have determined on whom we want a special examination and we have the material, it what to do with it. The men that sat around and discussed the problem know what they are talking about. They are men who in the profession are regarded in the top rank and when they lay down rules for the interpretation of electrocardiographic tracings and things are found which definitely point toward rejection, you can be pretty well assured that there is nothing in there that is of a controversial nature. We don't want to be in the position of rejecting an applicant for insurance and having him go to a well recommended and highly regarded private practitioner and have him come back with a perfect white wash and be in the position of having to defend a decision for rejection—so that if the recommendations governing electrocardiographic work definitely point toward rejection, there is complete and unanimous opinion

that because of the things which are recorded here none of the committee would recommend acceptance.

In the questionable group there is a little different problem. There are things in that group that from all experience published by clinicians, by hospitals and by others, if the abnormalities or the things found and noted in the questionable group mean that they carry with them a high mortality on a few cases studied, as we get more definite information on the questionable group, these cases would pass naturally into the unfavorable group, and, as time goes by, it seems to me that we can, within this Association, lay down rules for interpretation of electrocardiographic tracings in a way that has never been done before. I believe—and I have reason to believe—that clinical medicine will be the recipient of benefits from us which will take its place with blood pressure work, sphygmomanometry and other types of investigation.

I might say that in that group that are called questionable, the men that are handling these conditions feel that it is up to the individual decision of the company involved. I am not told to make this suggestion, but I would say this, that any smaller companies that have a tracing or an X-ray upon which they are in doubt as to just what it means, I personally and, I am sure, the other members of this group would be anxious to receive a film or tracing from any of the Medical Directors of smaller companies if they say, "Will you give us your opinion as to what this thing is?" If that work becomes burdensome—it probably won't—it will be necessary to set up machinery to handle it. If it isn't burdensome, I am sure all of us would welcome such requests. We will just have to try that out to see how it goes.

As far as X-ray interpretation is concerned, there is a little less unanimity of opinion. X-ray films are much more subject to opinion than electrocardiograph tracings, though, curiously enough, we know less about electrocardiography. In other words, what we do know about electrocardiography is more definite. The X-ray work is perhaps more open to opinion. It seems to me that the sensible way to handle that type of work is to realize that on any X-ray film we are getting more and better

information than we ever had before. If there is any controversy, then it seems to me—this is only personal opinion—that the fair thing to do is to give the applicant the break. Before he would have been accepted without any question. If there is anything that is less than definite, it seems to me that if we revert to our experience and various rating tables, we certainly are no worse off than we were some short time ago, and as our experience grows, we will know how to handle these things better.

The day before yesterday I sat at a table where one of the men was working on a report to be made at this Association a little bit later. He was bemoaning the fact that in this particular thing that he was studying he had no information on the impairment involved because we didn't accept that impairment. He didn't know what to say about it. We don't know whether that particular impairment is insurable or isn't because they are never accepted. The most important thing which is not being done now and which should be done is to follow up the applicants which are rejected on the basis of these special examinations. It is a very simple procedure. The only reason for including the Metropolitan's procedure in this report is simply because we have been doing it for some time. Our results have been uniformly good as far as being able to follow them is concerned, and it does record again a way which may suit your need or may not. The need for following rejected applicants is unquestionably there. Some of the cases which have been put up for investigation by us later would never have been found; never would we have known the subsequent developments of that case if we had not routinely followed the rejected applicants. We would not know that a case which one company examines today and rejects is subsequently examined by another company under a different name and a different date of birth. We would not know that that person subsequently within a few months applies for and receives disability. We would not bring out the things that we always felt but never have been able to prove about life insurance and sales in these modern days. I recommend most heartily, and this group feel the same way

about it, that immediately the individual companies that are doing any bulk of this work follow their rejected applicants so that they will know what happens to them. If we find something abnormal in the chest and are able to classify it—we'll say it is a healed tuberculosis—we don't know whether we are justified in rejecting that man or not—if we find various abnormalities that are questionable in the electrocardiographic tracing, the only way we will know the mortality of those questionable things is by following the rejected applicants. There is a scheme in the proceedings which is fairly simple and is very effective.

As I said, this is a very informal discussion of some suggestions made by this group.

DR. BIRCHARD—Mr. Chairman and Gentlemen: It will only take me one moment. In connection with this matter of electrocardiography in life insurance, to my mind there is absolutely no doubt but that the incorporation of this procedure in life insurance examinations will be comparable in benefit to the insurance companies with that which has been derived from the incorporation of blood pressure examinations. It may be more valuable; perhaps not quite so valuable—I don't know.

Now, as Dr. Fellows has reminded you, it is very important that the instrument makers should make good apparatus that can be used freely and without difficulty by people that have not got too much technical training. We have laid down some specifications as to the quality of instruments which appear to be reasonable. You will notice in this specification for electrocardiographic technique that we have stated that the deflection time of the instrument should be 1/50th of a second. In other words, the graph should be able to move through one centimeter on the film in 1/50th of a second. If you apply a voltage of 1/1000th of a volt, it should deflect one centimeter and should do it in 1/50th of a second. That probably is too easy for the instrument makers. It should probably do better than that.

The Cambridge Instrument Company have undertaken in the last few weeks to make some investigations, supervised by Dr.

Williams of Columbia—and, incidentally, Dr. Williams probably knows more about electrocardiographic apparatus than any other man living. Dr. Williams has agreed to assist the Cambridge Instrument Company in determining just how quickly the recording element of these instruments should move. Mr. Cruse of the instrument company told me last night that Dr. Williams is probably going to decide on 1/75th of a second, that the graph should move one centimeter in 1/75th of a second.

At the exhibit in the adjoining room, if you will take that portion of the graphs which are exhibited where you get a right angle deflection in the graph, that is the place where 1/1000th of one volt has been cut in the circuit. Now, the deflection should move promptly and precisely at practically right angles and take up its new position and stay there. You will notice in some of the graphs exhibited that it overshoots slightly. A little overshooting is permissable. Perhaps in some of them exhibited, as it comes to rest, instead of having a right angle turn it will have a slight curve. It is bound to have a slight curve. Those are the things which indicate to you whether that instrument is working well or whether it is not, and all the makers have been asked to exhibit graphs of that type which will show whether their instruments are in good working order or whether they are not in good working order.

It is stated in here that deflection should be made in 1/50th of a second. Probably that is too easy and should be of the order of 1/75th of a second.

Now then, with the valve tube instruments, the other type of instrument which is not quite so competent that is coming on the market and of which there are a couple exhibited upstairs, when the graph moves to one side, it will not stay there any longer than for a couple of seconds That does not interfere with the quality of the electrocardiogram. So that some of the graphs exhibited upstairs, while the graph moves properly to one side and gradually sags backwards, it is not a deffect and doesn't hurt its practical use in electrocardiography.

Other than that, these specifications which are being laid down to enable Medical Directors to know whether the instruments

which they will recommend to their examiners to buy are satisfactory are the biggest contribution, I think, that this organization can make at the present time. If we can see that all instrument makers put out good apparatus—and most of them do; some do not—we will have made a very big contribution to clinical medicine and one for which clinical medicine should be quite grateful.

DR. UNGERLEIDER—Dr. Fellows has given a very thorough exposition of the subject and I commend it to your serious consideration. You have heard Dr. Birchard's views on the electrocardiograph. My remarks will confine themselves to X-ray and they will be very brief.

What we are concerned with as life insurance medical men is a readable film. The heart shadow should be clearly and sharply outlined and the lung fields interpretable. The film should not be "muddy". It should be brilliant enough to enable the examiner to make a diagnosis of normal or abnormal findings.

I am not going to bore you with the technique of taking X-ray films but I will say that this group recommends to this Association the publishing of a small brochure which will contain the technique for taking satisfactory pictures.

In this brochure the suggestions of Dr. Fellows and Dr. Birchard, in regard to electrocardiography, should be incorporated. We suggest that this pamphlet be distributed to all the physicians on our electrocardiograph and X-ray list.

Furthermore, this group has available for any of the members of the Association who desire to install X-ray apparatus at their Home Offices, or elsewhere, data that will be of help to them in the selection of the proper equipment.

We all know that various manufacturers, whether they sell X-ray apparatus, automobiles or what-not, make extravagant claims. The data which we have and which is at your complete disposal will tell you exactly what you can hope to do with any apparatus.

One of the most irritating phases of our work is to receive a report of an X-ray film rather than the film itself. We

believe the X-ray film should be the property of the company that originally orders it, for two reasons, (1) for record purposes and (2) for their own interpretation. If the X-ray films are sent to the Home Office the company will be enabled to check up on the quality of work that is being done.

I know that the courts have held that X-ray films are the property of the rœntgenologist but if these men wish to do this work for us they will have to waive their right and forward the film to us for our records and interpretation.

In jumbo cases we believe that the X-ray film should not be supplanted by the orthodiagram. In these cases, when fluoroscopy is performed, an orthodiagram is desirable but it should never replace the X-ray. True, there are some clinicians whose orthodiagrams are very accurate and whose readings of lung pathology by the fluoroscope cannot be questioned, yet, if the X-ray film is waived we are losing a very desirable record. In these cases it is better to pay an additional fee and get the necessary requirement.

Finally, I believe it is an expensive procedure to photostat X-ray films. If a company desires details concerning the film, the original should be sent unless the inquiring company should ask only for cardiac measurements, or a statement concerning lung pathology.

To photostat a film and hope to show lung pathology is the height of folly.

DR. CHRISTIERNIN—At our meeting in Hartford last year, a considerable portion of the program was devoted to the requirements of the Joint Committee concerning electrocardiographic tracings and X-ray films. The papers dealt broadly with the general conditions of the work and a good foundation was laid for the future structure, but it was inevitable that problems and questions should arise later.

In that excellent paper that Dr. Birchard prepared, called "The Arterio-Sclerotic Heart", there appeared a paragraph which I have taken as a guide for a portion of this meeting. It reads as follows: "In the matter of heart size and its normal varia-

tions, there is awaiting action by this Association a very wonderful opportunity to serve scientific medicine: First, by laying down definite rules for radiographic examinations of hearts and educating the profession in their application. You have been largely responsible for accurate sphygmomanometry being widely obtainable; with less effort, rœntgenologists can be educated to make satisfactory plates of the heart and great vessels. Second, by statistically investigating the various measurement procedures and correlating the results with other anthropometric constants, such as body surface, weight, height, age, sex, race, etc., and eventually the mortalities obtaining in various groups. Third, by collaborating with the instrument makers towards the production of apparatus which will give the best possible results."

Taking these three suggestions in the order in which they come—first, "by laying down definite rules, etc."; early this year a group of our members who are particularly interested and showed the greatest activity at the last meeting were asked to meet in New York and they met several times and prepared suggestions and laid down definite rules, as Dr. Fellows has told you. We are hoping that these rules will be accepted generally, first by the members of this Association, and then the broadcasting of them through the special examiners and finally to the instrument makers.

Dr. Fellows—I merely want to close the discussion of the previous paper. The specifications for electrocardiographic and X-ray instruments which will do satisfactory work have been laid down by this group. If any of you men are contemplating the purchase of instruments, we would be glad and willing not only to let you know the companies that make such instruments, but to let you have those specifications which are scientifically exact and are subject to proof, so that you can determine whether you are buying something which will do the work or whether it won't.

Dr. Birchard spoke of an orthodiagram. I am glad he did because many of us probably don't know what it is. An orthodiagram is a tracing made on glass or paper of the direct exami-

nation of the heart as made by a fluoroscope, which is an instrument, of course, through which you look directly. In other words, if it is done properly, it may be more accurate than an X-ray. If it is done improperly, it may be much less accurate. We have seen in the past not infrequently jumbo risk cases where X-rays have not been ordered where the issuing company, the original company, has accepted a fluoroscopic examination of the chest as being the equivalent of an X-ray film of the chest. This we do not believe proper at this time.

DR. CHRISTIERNIN—Dr. Birchard's second suggestion, "By statistically investigating the various measurement procedures and correlating the results with other antropometric constants, etc.", Dr. Charles Nichols, of the Penn Mutual Life Insurance Company, has prepared a splendid paper on this phase of the subject. Dr. Nichols.

DETERMINATION OF CARDIAC HYPERTROPHY

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The most important single fact in the effort to determine the presence or absence of organic heart disease is the separation of the enlarged heart from one of normal size. The common etiological factors associated with cardiac enlargement are so well known that it would be superfluous to mention them, but a fact not so well appreciated, is, that all cardiac enlargement is not necessarily the result of work hypertrophy, a belief which has had universal acceptance. Recent advances in physiology have shown that prolonged overload or prolonged work, while important, is not the essential factor in cardiac hypertrophy but that stretching and injury to the heart muscle provide the necessary stimulus which eventually leads to enlargement. This stimulus may act over a comparatively short time and yet cardiac hypertrophy follow in its wake. Thus temporary elevations of blood pressure or a history of repeated elevations with subsequent normal findings assume a new significance. Inflammatory foci also result in exudate, dilatation and stretching of the muscle fibers. It is possible then to explain many cases of enlarged hearts, which, from an etiological standpoint are obscure, many arising on the basis of muscle injury with its temporary overload and stretching. Our knowledge, however, is still inadequate regarding the various anatomical processes which accompany hypertrophy but a weakened and dilated heart carrying out normal function is prone to enlargement under conditions which do not affect a normal organ. While this enlargement is accompanied by increased function, its reserve power is definitely diminished, so that sooner or later it fails to accomplish the task for which it was designed. So general is this consequence that we can always consider an enlarged heart

as a diseased heart. Thus a determination of what constitutes normal heart size becomes of paramount importance if we hope to separate the normal from the abnormal.

The shape of the heart of all mammals is approximately the same, being roughly conical with a wide base, rounded apex and slightly convex sides. It is however not geometrical since in human beings there is considerable flattening of the posterior wall. Before one attempts to measure the size of any given heart it is extremely important that he realize and take into consideration all the factors which are known to affect normal cardiac size and contour. The heart varies in size with the body weight, the height, the posture, the rate, phase of respiration, sex, age, nervous temperament and amount of physical exertion. Being an elastic organ its size varies with the volume of its contents, with venous and intrathoracic pressure and that elasticity of the muscle itself, which naturally varies from individual to individual, probably plays a part in its volume. Experience has thus taught us that there is no absolutely normal standard for the organs of the human body and we must concede the existence of more than one type of normal organ. Bodily habitus determines visceral form and function and the heart may best be studied by the variations in visceral morphology with bodily habitus. From this standpoint nearly all individuals, with slight variation, will fall into one of the following four major types:

The asthenic habitus presents a long, shallow and narrow thorax, with a heart which is long and narrow, being best described as "pearshaped". The upper half of the left border is almost a straight line and the contour of the right atrium and left ventricle show little convexity. The contour of the ascending aorta and aortic knob are only slightly prominent. The apex points downward and is often sharp.

The hyposthenic habitus reveals a thorax only moderately long but somewhat broader than the asthenic type. The heart still tends to be "pearshaped" but more convexity develops in the right atrium and left ventricular salient. The aortic knob becomes more prominent.

In the sthenic habitus the thorax is shorter and wider. The pulmonic fields are relatively wide at the base and narrow in the upper zone. The heart shape is "oval-globular" and the curves of the right atrium and left ventricle are only moderately convex.

The hypersthenic habitus reveals a short thorax with a large antero-posterior diameter. The pulmonic fields are very broad in their lower zones and the apices scarcely show above the clavicles. The heart is broad transversely, relatively short vertically, and generally oval. The curve of the ascending aorta is more pronounced and the upper mediastinal shadow relatively short but broad. The left ventricular curve shows marked convexity and the apex is broad and points outward. In many cases the apical curve cannot be made out at all.

Two individuals may possess the same sized heart but its diameters and appearance will vary with the type of thorax in which it is housed. A deep position of the diaphragm leads to a sinking of the apex medially and downward, the left border becomes straight and the heart appears smaller, without actually being smaller. With a high diaphragm the apex is displaced to the left and upward and both cardiac margins appear more laterally. The heart appears to be enlarged and at the same time its form is changed, suggesting hypertrophy and dilatation of the left ventricle. It follows, that according to the degree of elevation of the diaphragm we shall find various types of heart. How extreme this variation in the direction of the long axis of the heart may be, is shown by the angle between the tranverse and long diameters, which with different types of body build varies from 35° to 56°. Respiration has naturally the same effect on heart contour as varying positions of the diaphragm. In deep inspiration the cardiac silhouette grows narrower and longer, while at deep expiration it grows broader and globular.

It is quite generally appreciated that hypertrophy alone can cause comparatively little enlargement of the heart; the increased thickness of the heart wall which occurs in pure hypertrophy rarely amounts to more than a few millimeters and changes of

such degree are not easily recognized by any method. Recognizable cardiac enlargement means dilatation; all chronically enlarged hearts are dilated as well as hypertrophied. The increase in size is due very largely to dilatation, but the shape usually depends upon the amount of hypertrophy present. Thus dilatation gives predominately changes in size and hypertrophy, changes in shape. Well marked ventricular hypertrophy often gives no increase in measurements but the change can be recognized only by increase in the roundness of the left ventricle. In like manner the earliest enlargement of the right ventricle will be recognized by increased prominence of the conus pulmonalis and not by measurements. It is well to remember that in measuring the heart one should think in terms of centimeters and not millimeters.

The statement is often made that the heart grows larger with the increasing age of the individual. Naturally the heart increases with the mass of the body, then remains constant as long as body mass remains more or less fixed. That the onset of middle-age with its loss of physiologic elasticity of the large vessels results in hypertrophy of the left ventricle is in my opinion a doubtful assumption. A very extensive post-mortem experience has convinced me that the heart of the older individual is a small heart, that if the ageing process is a gradual one the heart by a slow decrease in its capillary bed becomes smaller. If it is not small but enlarged, there can usually be found a definite etiological factor to account for it.

With this brief and incomplete discussion of the fact that the size of the heart is physiologically dependent on a number of body properties, that an appreciation of them is essential to any understanding of the tremendous variation in the size and form of the normal heart, we can proceed to an evaluation of the various methods used to determine heart size.

In our zeal to apply mathematical accuracy we are prone to forget the great value that lies in a carefully taken personal history and a properly performed physical examination. The one clinical method of practical value is the location of the apex beat. It is approximate, and often inapplicable to obese

and heavy chested individuals, but it suffices in young subjects and adults of normal build. Certain precautions must be observed. Simple inspection will often reveal the location of the apex beat, especially if the chest is bared in the proper light. Sometimes exercise will develop a visible beat not seen before. Palpation however is by far the most exact method of locating the apex thrust. We are seeking not the outermost point at which the cardiac impulse can be appreciated, but by feeling along the interspace in the nipple region we seek for the left-most point on the chest where a distinct lift is imparted to the palpating finger tip. The heart should be beating quietly and at a normal rate as in rapid heart action the apex beat is much diffused. The purpose in ascertaining the position of the apex is to relate it in some manner with definite anatomical land marks. The usual ones being the nipple and the mid-clavicular line, subscribing to the idea that in long thin subjects the left border of the heart approaches the sternum as do also the nipple and mid-clavicular lines, while in short stocky subjects the reverse is true. The apex of the heart is a truncate cone and the anatomical border may be 1 to 2 cm. to its left. In about one-third of all cases the apex will coincide exactly with the anatomical left border. In healthy subjects the apex beat varies from 2½ to 4½ inches from the mid-line, varying with the size of the individual. It may be over four inches out in a large man and be normal, while in a smaller individual it may be 3½ inches out and yet be enlarged. The mid-clavicular line is considered to bear a fairly constant relation to the size of the chest, whereas the nipple is often considered a variable factor. In my opinion the nipple is an excellent anatomical landmark in adult males, and it will be exceedingly rare to find the apex beat of a normal heart beyond the nipple. The same principle is of course applicable to the mid-clavicular line and 95 per cent. of all normal hearts lie within this line. In enlargement of the outflow tract of the right ventricle the apex and left border of the heart may be normally located but the enlargement occurring upward and to the left must be discovered by percussion above the lower portion of the sternum,

when increased flatness, due to bulging forward of the anterior right ventricular wall will be a valuable sign. Too much emphasis cannot be placed upon the value of palpation as a means of estimating heart size. The palm of the hand must be pressed firmly over the precordia in the nipple region before more exact localization is attempted with the finger tip. With the finger tip you are seeking the out-most point of forward thrust at right angles to the chest wall, not an oblique thrust. This method should give accurate results where applicable in over 90% of the cases. Those skilled in percussion can usually locate the left heart border accurately, as checked by orthodiagraphy but they will practically always be inside the right border. So few are skilled in percussion that it has seemed to me of little value as usually practiced. If palpation fails, percussion is more apt to do so and we must rely upon the various radiological methods for studying cardiac contour and size.

Upon the rœntgenologic estimation of heart size there are greater differences of opinion than upon any other subject which has received an equal amount of attention. A perusal of the foreign and domestic literature upon this subject during the past fifteen years would leave the reviewer in a maze of indecision; yet it is possible to evaluate some of these methods and lay a temporary ground work upon which we in our special field of medicine can build for the future. In the early days we expected, without reason, that the rœntgen-ray would distinguish infallibly between a heart which is just beginning to enlarge as a result of disease and one which is normal. This was obviously soon found impossible. The border land between health and disease cannot be demarcated by any sharply drawn line. Even the magic of the rœntgen-ray is bound to leave a zone of uncertainty.

In the rœntgenologic examination of the heart three methods are available: Fluoroscopy, orthodiagraphy and telerœntgenography. Fluoroscopy cannot be used for exact estimation of the size of the cardiac shadow, since the image which appears on the screen is magnified by the divergence of the X-ray emanat-

ing from the tube which is close to the patient and screen. It should be used to study the contour of the heart and each individual chamber, turning the patient in all positions as the examination proceeds. No other single procedure is capable of giving as much useful information as the fluoroscopic study, which can be completed in 3 to 4 minutes. A knowledge of the normal configuration of the heart and its variations with various types of bodily habitus is absolutely essential. To gain this knowledge one should have fluoroscoped at least 500 normal hearts. The first changes produced in the heart by hypertrophy are changes in form involving one or more cardiac chambers. These changes in form occur and can be recognized long before any measurement of the heart would suggest enlargement. A systolic murmur at the apex without any other positive findings might be considered accidental, but the demonstration of a left auricle protruding into the retro-cardiac space would give the case an entirely different significance and a much higher mortality. It is my conviction that the fluoroscopic study alone would permit the separation of applicants with systolic apical murmurs into two distinct groups, one giving a very favorable mortality and another giving a very unfavorable one. Space does not permit of a lengthy discussion, which this phase of the subject deserves. In the well equipped home office of the various companies there should be an experiencd fluoroscopist and in all large centers the companies should have men of sufficient training and experience to render competent decisions upon cardiac size. These men exist, they are available but they are not found among the ranks of the average rœntgenologist or even the very experienced cardiologist. American medicine has fallen far behind in this phase of cardiac study.

Teleradiography involves taking a radiogram of the chest with the film at a distance of two meters or more from the tube. This gives the shadow of the heart with its curves and angles, constituting a real projection of the organ. At this distance the rays are nearly parallel and the distortion from divergence of rays is slight, though it cannot be ignored. With more powerful apparatus which is coming into use greater film distance is

possible and distortion from divergent rays can be practically eliminated. A rigid technique is essential to the production of accurate films. The applicant must be carefully centered in order to obviate distortion from rotation and the tube exactly centered at the level of the sixth thoracic vertebra. The axis of the tube should be parallel to the plane of the film and the anterior chest wall of the applicant. If maximum size of the heart is desired, exposure should be long enough to include diastole of the ventricles. Exposure time should then not be longer than one-half second. It is necessary also to watch respiration closely during exposure, for at deep inspiration the cardiac silhouette grows narrower and longer, while at deep expiration it grows broader and globular. The exposure is preferably made with the breath held during the median phase of quiet superficial expiration. It is possible to use a timer actuated by a mechanism set off by the pulse wave and obtain exposures synchronous with the diastolic phase of the heart cycle. Such complicated and expensive technique is entirely unnecessary. The average excursion of the left heart border during a cardiac cycle at normal rate is between two and three millimeters. Slower hearts with a rate of 60 or under would have a maximum excursion of possibly 4 millimeters. In the case of heart block with a rate of 30 or under, relatively long diastole, the excursion of the left border may be as much as 8 to 10 millimeters. Now for practical purposes we are not concerned with minor changes of 2 to 3 millimeters, so the excursion of the heart can be neglected. With modern apparatus we can use exposure time from 1/30 to 1/120 of a second and thereby ignore the excursion of the heart, the resulting picture may represent the heart either in systole or diastole or at some phase between. With fast exposure we produce clear, sharp-cut pictures, which can depict calcifications in the aorta, pericardium and possibly the coronary arteries. They also produce excellent lung detail and clearly define the main branches of the pulmonary arteries. This technique should be definitely demanded by all companies, since it will greatly improve cardiac selection.

While this method is not ideal it does represent more or less exactly the projection of the cardiac area in one plane only. The heart being projected in only one direction of the rays, it is a two dimensional shadow of a three dimensional body. Having thus obtained a silhouette of the heart, its size can be measured by drawing diameters between given points on its contour, or by a planimetric determination of the area of the silhouette. The latter method should not be applied to films, since the necessity of arbitrarily completing the upper and lower borders, injects a subjective error of considerable magnitude. The error in surface area measurements is smaller in ortho-diagrams, altho still a serious one. It gives better results in the small, asthenic type of heart, but in the stout group, where most needed, the error is considerable.

Various authors have published a large number of heart diameters which in their entirety give an approximate measure of the heart size. The use of these diameters has not proved practical, rather, they tend to confusion, so we seek to accomplish our purpose as far as possible with only one measurement. This measurement is the transverse and consists of two semi-diameters; they are traced from a vertical line in the mid-line of the body to the farthest points of the heart shadow on the right and left side. These right and left median distances have a fairly constant relation of 1:2. The transverse diameter thus passes through the right auricle, right ventricle and left ventricle. In normal adults it averages about 12 cm. with a plus or minus normal average variation of 3 cm. It must be remembered that the transverse diameter indicates the width of the image of the heart but does not express the width of the organ. The reason for this is clear when we realize that the heart has an oblique position within the chest and has a direction from the right side and above downward and to the left side as well as from behind forward; therefore the transverse diameter of the heart does not correspond to the transverse diameter of the image.

The relation of the transverse diameter of the heart to the largest diameter of the thorax, the so-called cardio-thoracic

ratio, has been the widest used application of this measure. It has been assumed that when the ratio exceeds 50 percent. (some consider 55 or even 60 per cent. the upper limit) the heart is enlarged. This is obviously a very crude method and subject to large errors. It can be conservatively said that the use of this method will fail to detect at least 30 per cent. of enlarged hearts. The inaccuracy lies in the generally accepted fact that the size and weight of the normal heart, even when correlated with other body measurements, show such variations that its latitudes cannot be exactly delineated. Variations in bodily habitus may cause the image of the heart to appear large or small without the volume being really modified. The percentage of error will be greater with the larger hearts, since the rays outlining a large heart are more divergent than those outlining a small one. The transverse diameter of a small heart may be only one-half of a centimeter larger than the actual measurement checked by autopsy, while the transverse diameter of the shadow of a large heart may exceed the actual by one to two centimeters. Often the long diameter of the heart can be used to give some information, especially when the apex is visible. This diameter runs from the auriculo-aortic junction to the apex of the left ventricle and is normally greater than the transverse diameter by .5 to 2 centimeters. If it is shorter than the transverse diameter the heart is either abnormally horizontal in position or is too wide. If 3 or more centimeters longer than the tranverse, the heart must be abnormally vertical in position or else too long. Pure hypertrophy of the left ventricle finds its expression in elongation of the left border. The apex is slightly lowered but it is not pushed backward so that the transverse diameter remains normal. Pure hypertrophy of the right ventricle finds its earliest expression in a prominence of the conus pulmonalis when due to out-flow tract enlargement. Thus changes in contour give the clue and these changes in contour of the various salients of the heart border must be carefully studied.

In spite of its obvious defects, life insurance is more and more dependent upon the long distance film for its estimate of heart size. Herein lies a real opportunity for us to make a

new and valuable contribution to medical science. At present the writer knows of no accurate figures for transverse heart size taken from the teleradiogram, therefore, we must supply them from the tremendous number of films which we will collectively receive. But we should first demand technically correct films showing contrast and clearly defined outlines, with penetration and intensity adapted to the weight and thoracic girth. We should emphasize and point out the great need for symmetrical projection. We should then correlate the transverse diameter with body weight, age, height, sex, shape of the chest and possibly the position of the diaphragm. With this vast statistical data, we will soon develop some accurate ideas upon the normal size of the heart. The need for this is especially urgent in the age groups from 40 to 60 years.

All companies in their home offices should have trained men who know the general principles of radiological projection. These men should interpret the films and the one great advantage the film has over any other method, is, that you can at once recognize improper centering and abnormal rotation. You cannot correct it but you can at least recognize it and thus avoid bad errors which are being made daily on submitted films.

Recent attempts have been made to correlate the cardiovascular shadow in the left oblique view with the chest cage, thus permitting comparative mensuration of the two. Granting that correlation between the heart and thorax in the postero-anterior position is grossly inaccurate, it seems difficult to believe that the left oblique view would lend itself to anthropometric analysis. The excursion of the heart in the left oblique view at between 45° and 55° is much greater than in the postero-anterior view, probably between 4 and 6 millimeters, so measurements here, would, to be accurate require synchronization of exposure and heart cycle, a complicated and unnecessary procedure. Short exposures with higher milliamperage give excellent results in oblique views. Working with intact hearts at autopsy we have as yet been unable to satisfy ourselves that the septum of the heart can be located with a degree of accuracy sufficient to warrant its use as an anatomical landmark in apply-

ing mensuration to left oblique films. Judgment, however, should for the present be reserved. Since we are dependent upon films coming from laboratories of varying degrees of merit, it would seem wiser at present to concentrate on postero-anterior positions; rather than the left oblique which requires a rigid technique to produce suitable films.

AVERAGE, MAXIMAL AND MINIMAL VALUES OF TELEORŒNTGENOGRAMS (2 METERS) STANDING, ARRANGED ACCORDING TO AGE (AFTER HAMMER)

Group	Mr.	Ml.	Tr.	
	2.7	6.1	11.4	minimal
1. (18)19–21 years	4.6	8.5	13.1	average
	6.3	11.0	15.6	maximal
	2.8	7.7	11.8	minimal
2. 22–24 years	4.4	9.0	13.4	average
	5.8	11.6	15.2	maximal
	3.6	7.2	11.8	minimal
3. 25–29 years	4.8	8.6	13.4	average
	6.9	9.7	14.7	maximal
	3.9	7.6	12.6	minimal
4. 30–34 years	4.9	8.8	13.8	average
	5.9	10.6	15.1	maximal
	3.5	8.1	12.7	minimal
5. 35–39 years	4.9	9.2	14.1	average
	6.4	11.0	14.8	maximal
	3.9	7.4	15.2	minimal
6. 40–45 years	5.7	9.6	15.3	average
	7.9	11.3	15.5	maximal

Orthodiagraphy involves directing a bundle of central rays vertical to the frontal plane of the body and serially inscribing the configuration of the heart upon a fixed fluoroscent screen. The rays being vertical and non-divergent, it produces a true-sized silhouette of the heart in a single plane. Its exactness is generally recognized and in practiced hands is considered the method of choice. The procedure may be carried out in either the horizontal, sitting or vertical positions and the horizontal seems to offer some advantages, in that the heart shows less variation in size, the patient is better immobilized and the apex

more often visualized. It is however the vertical position which is commonly used, since it requires less complicated apparatus. In changing from the horizontal to the vertical position there is a definite decrease in the area of the heart shadow. The heart volume decreasing about 25 per cent. and the area of cardiac outline about 12 per cent. This accounts for the variation found in many published tables. No matter what method is employed the patient must be immobilized, the slightest movements being markedly increased on the screen. The tracing should be inscribed during diastole and in the phase of quiet superficial inspiration. Subjective mistakes occurring during this procedure are small, being not more than 3 to 5 mm.; changes of a few millimeters have no practical value as only changes exceeding 0.5—1.0 cm. may be taken into consideration. Again let it be emphasized that in measuring the heart one should think in terms of centimeters and not millimeters.

The size of the silhouette thus obtained may be expressed in square measure as area or in linear measure by drawing in certain diameters. Many measurements have been proposed, many of them complicated and impossible to accurately delineate, therefore, we shall confine our discussion to those which seem to have a practical application. The transverse diameter is obtained by adding together the two horizontal diameters drawn from the mid-line to the most distant points on the right and left borders. The area is obtained by completing arbitrarily the upper and lower boundaries of the heart by curved lines and then determining the contained area by means of a planimeter. The great objection to area as an index is the personal factor involved in completing the boundaries and the time involved in measurement. Area is a less valuable measurement than transverse diameter, since the variation between different observers would be greater than in the case of linear measurement. For those who wish to use area of the orthodiagram as an index of heart size the tables of Eyster and Hodges should be employed. They have used height and weight as variables, believing that these correlate better with the size of the heart than any other measurements. In the hands of experienced

observers this method has real merit but it suffers from one great defect, in that these tables were derived from healthy young adult males whose age limits were narrow. Applying these tables to the age group 35 to 55 years, where it is most needed, the prediction will be much less successful.

The transverse diameter of the silhouette of the frontal plane orthodiagram using age, height and weight as variables represents the most valuable method for differentiation that we can apply. Eyster calculated the coefficients of correlation between various heart and body measurements. Thus for transverse diameter he has published tables which are widely used. In a recent personal communication Eyster states he has verified these tables by more elaborate recent studies on several hundred groups of normals and found that they were very substantially correct. We are still badly in need of measurements taken from higher age groups and this defect we are in a position to correct. The error involved here must be between 10 and 20 per cent. when considered in terms of volume. Thus a variation of plus or minus 15 per cent. from the predicted normal would be the best method of applying these data. They will be more accurate for those who fall into the ideal weight for height group. In those too tall or too short for their weight the error will be greater. They probably represent the best prediction of transverse diameter that we can use but measurements expressed in millimeters connote accuracy which may be misleading. Simple inspection of the orthodiagram will enable an experienced worker to differentiate the large majority of enlarged hearts since changes in contour are more important often than measurements. Thus our measurements must be used only as they relate to the clinical picture as a whole. A departure from predicted normal would be much more significant in one with a history of rheumatism or a slightly elevated blood pressure than would be the case in an applicant who was apparently normal. Also past histories of elevated blood pressure or the finding of a temporary elevation would make us more skeptical in evaluating departures from the so-called normal. Recent foreign work of a very reliable nature tends to show that the dimensions of normal

hearts with the same height and weight fluctuate considerably, namely within the limits of one centimeter or more. With due consideration of this fact we should not attempt to apply any rule-of-thumb measurements but to correlate all our data in reaching a decision. It requires nice clinical judgment.

One of the best tables based on orthodiagraphic work is that of Rautmann who studied a large number of normal individuals and correlated the size of the heart with the various constitutions of the carrier. By the aid of regression cœfficients for body size, body weight, circumference of the chest and size of the heart, he was able to prepare a correction table in reference to the body weight and the circumference of the chest, in which it is stated in how far the transverse diameter of the heart is changed on the average when the body weight or circumference of the chest deviates from the given average values upward or downward.

TABLE I—DEFINITION TABLE FOR TRANSVERSE DIAMETER OF HEART

Ht. cm.	Wt. kg.	Chest Circum cm.	Trans. Diam. Heart cm.	Ht. cm.	Wt. kg.	Chest Circum cm.	Trans. Diam. Heart cm.
150	51.0	81.0	12.8	173	67.1	86.8	13.4
151	51.7	81.3	12.8	174	67.8	87.0	13.4
152	52.4	81.5	12.9	175	68.5	87.3	13.4
153	53.1	81.8	12.9	176	69.2	87.5	13.4
154	53.8	82.0	12.9	177	69.9	87.8	13.5
155	54.5	82.3	12.9	178	70.6	88.0	13.5
156	55.2	82.5	13.0	179	71.3	88.3	13.5
157	55.9	82.8	13.0	180	72.0	88.5	13.5
158	56.6	83.0	13.0	181	72.7	88.8	13.6
159	57.3	83.3	13.0	182	73.4	89.0	13.6
160	58.0	83.5	13.1	183	74.1	89.3	13.6
161	58.7	83.8	13.1	184	74.8	89.5	13.6
162	59.4	84.0	13.1	185	75.5	89.8	13.7
163	60.1	84.3	13.1	186	76.2	90.0	13.7
164	60.8	84.5	13.2	187	76.9	90.3	13.7
165	61.5	84.8	13.2	188	77.6	90.5	13.7
166	62.2	85.0	13.2	189	78.3	90.8	13.8
167	62.9	85.3	13.2	190	79.0	91.0	13.8
168	63.6	85.5	13.3	191	79.7	91.3	13.8
169	64.3	85.8	13.3	192	80.4	91.5	13.8
170	65.0	86.0	13.3	193	81.1	91.8	13.9
171	65.7	86.3	13.3	194	81.8	92.0	13.9
172	66.4	86.5	13.4	195	82.5	92.3	13.9

TABLE II—CORRECTION TABLE FOR WEIGHT VARIATIONS WHEN WEIGHT VARIES FROM AVERAGE VALUE

by	1.00– 2.25kg.	changes	heart	size	by	0.1cm.
"	2.25– 3.75kg.	"	"	"	"	0.2cm.
"	3.75– 5.25kg.	"	"	"	"	0.3cm.
"	5.25– 6.75kg.	"	"	"	"	0.4cm.
"	6.75– 8.25kg.	"	"	"	"	0.5cm.
"	8.25– 9.75kg.	"	"	"	"	0.6cm.
"	9.75–11.25kg.	"	"	"	"	0.7cm.
"	11.25–13.25kg.	"	"	"	"	0.8cm.
"	13.25–14.75kg.	"	"	"	"	0.9cm.
"	14.75–16.25kg.	"	"	"	"	1.0cm.
"	16.25–17.75kg.	"	"	"	"	1.1cm.
"	17.75–19.25kg.	"	"	"	"	1.2cm.
"	19.25–20.00kg.	"	"	"	"	1.3cm.

TABLE III—CORRECTION TABLE FOR CHEST CIRCUMFERENCE WHEN CHEST CIRCUMFERENCE VARIES FROM AVERAGE VALUE

by	1.00– 2.25cm.	changes	heart	size	by	0.1cm.
"	2.25– 4.25cm.	"	"	"	"	0.2cm.
"	4.25– 5.75cm.	"	"	"	"	0.3cm.
"	5.75– 7.25cm.	"	"	"	"	0.4cm.
"	7.25– 9.25cm.	"	"	"	"	0.5cm.
"	9.25–10.75cm.	"	"	"	"	0.6cm.
"	10.75–12.25cm.	"	"	"	"	0.7cm.
"	12.25–14.25cm.	"	"	"	"	0.8cm.

May there be any relation between the size of the heart and its efficiency as a pump? Is it conceivable that the heart may be too large or too small for the individual who carries it? The efficiency of the heart should no doubt be measured in terms of blood-flow and the character of its pulsations as well as its relative size should not be used as a measure of the condition of the muscle. There are no doubt individuals who have hearts relatively too large for their body mass. The only explanation for this, is that a familial, constitutional factor is at work. It can be traced in members of the same families. On the contrary the small, pendulous heart may be very efficient as a pump, since many of these individuals are excellent athletes. However, there is a close relationship between slender build and efficiency of the cardio-vascular system, since nearly 70 per cent. of the effort-syndrome group are too tall for their weight and

have relatively small hearts. Here again other factors than constitutional inferiority may also play a part. It would seem impossible in the light of our present knowledge to give a definite answer to the question whether a given heart may be normally too large or small for its carrier. For the present we should consider those values for heart size as normal which are found most frequently in healthy individuals and only rarely found in individuals suffering from various affections.

No consideration of the heart is complete without a discussion of the aorta. Much of the elaborate rœntgenographic studies in the past have been worthless, due to an insufficient appreciation of the anatomic variations in this vessel. The most important factors in this variation are the age of the individual and the varying spatial relations of the thoracic cage. Postmortem studies have shown that the circumference of the aorta increases progressively from youth to old age. When the diaphragm is high the aorta is spread and becomes more prominent to right and left. In low positions of the diaphragm the aortic shadow is narrower, with but slight bulging to right and left. Due to loss of elasticity the aorta uncoils with advancing years, enlongates, becomes spiral and appears prominently and high to the left. Thus fluoroscopy again becomes the most important method that can be applied to its study but its various anatomical positions must be thoroughly understood. The individual must be studied in all positions, since tortuosity, dilatation and elongation may only be perceived in a lateral or oblique view. In the normal individual below the age of 45 years, the ascending aorta can be seen in all views and, when viewed in the frontal plane, lies behind and somewhat to the left of the sternum, from the level of the first rib to approximately the third. The right border of the shadow includes the superior vena cava and pulsations to the right of the sternum are rarely seen in a normal vessel. The thoracic aorta cannot normally be visualized before the age of 45 to 50 years. After the age of 45 certain changes begin to progressively appear due to loss of elasticity, it elongates and increases in density, spreads out laterally and pulsations become visible to the right

of the sternum. Due to increased density the thoracic aorta can be seen in the lateral views and often in the frontal. After middle life the extent of these changes depends upon the tempo of the degenerative processes which are occurring. When these changes are found in the thirties or early forties, it will be safe to presume some infectious or degenerative process is at work. After middle life, considerable clinical judgement based on wide experience is necessary in deciding whether the observed changes are in excess of what is to be expected at that age.

The most frequent measurement applied to radiograms is that of the shadow made by the great vessels, the so-called vascular pedicle. This shadow includes the venous channels on the right, the mediastinum and the varying anatomical positions of the aorta. It may be considered of some interest but of decidedly limited value. The measurement is usually made at the level of the 2d interspace and varies from five to seven centimeters in the adult. The measurement not only varies with pathological changes but also with normal variation in the spatial relation of the thorax. It is a most variable factor.

The method of Kreuzfuchs' is simple but requires barium visualization of the aortic bed of the esophagus. In the antero-posterior position, it is a measure of the length of a horizontal line drawn from the most prominent bulge of the aorta at the left of the sternum to the shadow of the barium-filled esophagus. The upper limit of normal for this portion of the aorta is usually stated as 2.8 cm. It measures the aorta at the isthmus and not at the ventricular portion where it is most often of importance. All other measurements which have been applied to the aorta are quite unsatisfactory.

My measurements for the ascending aorta, taken from a large series of autopsied cases, average between 3 and 4 cm. for tranverse diameter. After death the aorta is empty, sags somewhat, becoming elliptical. This increases the transverse diameter over what it would be in life, when the vessel is truly spherical, by approximately 1 cm. The true transverse diameter of the normal ascending aorta in life lies between 2 and 3 cm. When at autopsy we find an aorta measuring 4 cm. or

more, we can confidently predict that it is the seat of inflammatory or degenerative disease. These measurements are taken from the supra-cardiac portion of the ascending aorta, a measurement difficult or impossible to accurately make in life.

Attempts to correlate the measurements of the aortic arch with certain diameters of the chest in the left oblique position have proved of some value but need confirmation. Teleradiograms taken in the left oblique at 45° and using rapid exposures are of much more value, since they will reveal structural changes, such as calcification, filling defects, dilatation, diffuse stretching and thinning. We will increasingly demand and need the valuable information which these oblique films can give us.

There is at present no consensus of opinion as to cardiac measurements. One group of careful workers will discard all mensuration and depend entirely upon clinical judgment. Another group will attempt to measure the heart and decide enlargement in terms of a few millimeters. Somewhere between these two extremes lies the truth. We can truthfully say that measurements have a real value but they must be interpreted very freely. We cannot fit our cases into narrow, standard limits but if the measurements are properly correlated with the whole picture which the individual presents, they have great value. For the present we should make increasing use of the film, since it has one great advantage over other methods. If we understand radiological projection we will be able to tell at once whether the applicant was properly centered, whether he was rotated, or if other mistakes in technique were made; we cannot correct these mistakes but if we recognize them great errors can be avoided.

This represents but a cursory review of a subject about which volumes have been written, to delve into the technical aspects would carry us far beyond our allotted time and serve no useful purpose. Our desire has been to point out only the main considerations which underlie this work and to suggest methods whereby insurance medicine can best profit from these various procedures.

DR. NICHOLS—In presenting this communication our object was not to place before you a simple, "fool-proof" method for determining normal cardiac size, since it is our belief that no such method exists. We did hope, however, to point out a few of the underlying principles which should guide one in carrying out this work and, thus, emphasis has been placed upon the great variations in size and shape which exist in the normal heart. That these variations can be limited to a certain extent by relating the size of the heart to other body measurements, notably the total surface area of the body, is fully realized but the problem is still far from a satisfactory solution.

While it is quite convenient to be able to put down certain figures and measurements in regard to a given heart, we often have no means whereby we may know what the size of that particular heart ought to be in the absence of disease. Physicians have always been more prone to diagnose and treat disease in preference to making the basic anthropologic and statistical studies that would make it possible to say when normality ends and disease begins. This problem of supplying "standards of normal" for heart size awaits us and with our vast statistical data much can be accomplished if this data is properly correlated with all the various constituents of the carriers.

Emphasis cannot be too strongly placed upon the importance of studying changes in the shape of the cardiac silhouette and the great value that lies in early recognition of abnormalities in size and position of the individual chambers. These factors are to me more valuable than any measurements that can be made. Certainly the Danzer ratio is of little value, since a heart, originally very small, can enlarge enormously before it exceeds the maximum dimension in its group and a heart naturally large passes the normal maximum with the slightest increase in size.

We don't want to take any undesirable risks, but at the same time we don't want to place obstacles in the way of our companies taking desirable risks, and unless we bring to bear all of our experience and judgment and clinical acumen upon this subject, we are going to make mistakes. It is no easy matter.

DR. CHRISTIERNIN—The discussion of Dr. Nichol's paper will be opened by Dr. Hugo Rœsler, who is Associate Professor of Rœntgenology and Associate Professor of Medicine at Temple University Medical School. Dr. Rœsler is a graduate of the University of Vienna. He spent six years at the Vienna Heart Hospital and five years as Assistant Rœntgenologist at the Vienna Polyclinic Hospital. Dr. Rœsler.

DR. ROESLER—In order to diagnose and determine the degree of abnormality of the shape, size and action of the heart and the great vessels, one must know what is normal. This draws attention to the question of how large the range of variation of the normal is and what we should call the upper and lower limit of the normal.

We are still far away from being able to express in mathematical terms our problems, thus fulfilling the definition: Science consists of the power of prevision based on quantitative knowledge.

I. The weight of the heart can be deduced from the weight of the body (and that of the kidneys) by means of a linear equation with an average error of about 8%. Extensive studies have not been made to prove or disprove that this holds true for people who are highly overweight or highly underweight.

Purely from the anatomical serial examinations which have been made one gains the impression that the heart weight increases in the fourth and fifth decade irrespective of the body weight. However, one must consider that except for the statistics of Smith from the Mayo Clinic, the blood pressure findings were not taken into consideration. It seems very probable that this increase in weight is caused by the presence of a number of hearts which had reacted to hypertension.

The constitutional factor plays a rôle. People in whom the mass of the chest and abdomen is greater than that of the extremities have an average heart weight which is 10% greater than that of people in whom this proportion is reversed. From this fact, it can be deduced that the measurement of the chest circumference must bear an important relationship to the determination of heart size.

The aorta increases continuously in its longitudinal and transverse diameter throughout life. It is, therefore, logical to expect an increased curvature and increased size of the aorta in the x-ray picture with increasing age.

There are two anatomical types of the descending thoracic aorta: The pre-vertebral type and the para-vertebral type. In the pre-vertebral type, the aortic knob shows clearly and the total width of the vessel shadow is smaller. In the latter type the shadow of the descending aorta is seen for a longer distance and thus the total width of the vessel shadow is larger.

The circumference of the aorta shows a rather wide range of variation for normal people as has been demonstrated in extensive anatomical studies, especially on German soldiers during the war. This fact must always be remembered before passing judgment on a small or wide aorta.

If either ventricle or both of them undergo hypertrophy, the process will affect first the outflow tract. Thus, in the case of the right ventricle, the regino of the conus arteriosus will hypertrophy; the level of the pulmonary valves moves upward thus becoming superior to its normal position. The anterior wall of the right ventricle moves forward. The result of this change shows in the x-ray picture as a prominence of the conus arteriosus, which can best be seen in the anterior and right anterior oblique position. The shape of the left heart contour changes but the measurement of the heart still gives normal figures. Clinically, percussion of the lower half of the sternum reveals increased dullness. In the case of the left ventricle, the anterior wall of this ventricle and the adjacent parts of the septum below the aortic valves lengthen and increase in thickness. The level of the aortic valves, therefore, rises cephalad above the level of the mitral valves. No typical x-ray picture is known for this stage and it is uncertain whether a clinical diagnosis of left ventricular hypertrophy by palpating the apex beat can be made at this time. During the second stage, hypertrophy and dilatation of both ventricles or either ventricle occurs. The inflow tract is thus affected in such a way that in the case of the right ventricle, the space from the tricuspid valves to the apical por-

tion increases, and in the case of the left ventricle, the space from the mitral valves to the apical portion increases. This increase results in a globular enlargement and a uniform thickening of the ventricular wall which may be seen in the x-ray silhouette. If the condition affects the right side of the heart, a predominating increase in the transverse diameter of the heart and a straightening of the waist of the heart occurs. If it affects the left side of the heart, however, the silhouette shows a lengthening and increased rounding of the left ventricular contour and a deepening of the waist of the heart.

If one compares the apical region of the x-ray silhouette with the clinical apical forward thrust, one will find a coincidence in about 40% of the cases. In the rest of the cases, the clinical apex beat will be found three times more often outside than inside of the x-ray silhouette. For this reason, we can see why over-estimation of the clinical heart size occurs more often than under-estimation.

The right heart border is deeper located in the chest than the left heart border. The average distance from the skin to the outermost right heart border is 4 to 5¾ cm. and to the outermost left heart border 3 to 4½ cm. The right auricular wall, furthermore, as compared with the more medially situated right ventricular wall, is thin. These two facts explain why percussion of the right heart border will always fall considerably inside of the right x-ray cardiac silhouette. The normal aorta is too far away from the anterior chest wall to give any dullness but if percussion reveals dullness of the upper half of the sternum, it indicates, unless other mediastinal conditions are present, widening of the aorta or abnormal tortuosity. Either tortuosity or widening of the aorta bring it closer to the ventral chest wall.

II. A teleroentgenogram is important as a record, for it enables us to judge at a glance whether the patient was rotated slightly and reveals clearly calcification in the aortic wall or in the pericardium. As compared with fluoroscopy and orthodiagraphy, it has a disadvantage in that the apical region can never be so well defined. The lower contour of the heart shadow cannot be well distinguished and the region of the auricular ap-

pendix which separates the pulsations of the pulmonary artery and the left ventricle cannot be located. These three facts increase the degree of unexactness of area measurement in the teleroentgenogram.

The difficulty of outlining exactly the apical contour may be traced to two causes. Normally a kind of ligament extends from the pericardium to the mediastinal and diaphragmatic pleura and in the same region there is a certain amount of fat. This formation as well as the diaphragm cast about the same density of shadow as the heart does. In fluoroscopy, the heart can be separated from these formations on account of its continual motion.

Orthodiagraphy is a subjective method and cannot be well done without having had special instruction by an experienced man and without considerable practice.

Different standards of normal size should be used for orthodiagraphy and teleroentgenography respectively because even in the 7-foot film, the rays are not parallel and the difference of area measurement for these two methods are about 4 to 10%, teleroentgenography giving larger figures.

The degree of magnification depends upon the distance of the heart from the film and this is influenced by the chest type and by the size of the heart itself. If the target film distance is 2 meters and the distance of the main heart diameter from the plane of the film 6 cm., the magnification is 1.25%. If the distance is 9.1 cm., the magnification is 4.7%. In order to produce a constant error of 5%, for both hearts, we would have to choose for the 6 cm. depth a target film distance of 1.25 meters and for the 9 cm. depth a target distance of 1.9 meters.

An error of 5 mm. in the measurement of the transverse diameter of a heart of average size gives an error of about 15% in volume.

It has been mentioned already that the right heart contour is deeper in the chest than the left one. It may be added that in 45 degree left anterior oblique position, which has been recently used for purposes of measurement, the right sternal border of

the heart is several centimeters further away from the plane of the film than the left vertebral heart contour.

The main heart axis has a direction from the right side to the left and from behind forwards. In order to examine the heart parallel to this main axis, one would have to rotate it into 25 to 30 degrees right anterior oblique position.

The study of heart pulsations by fluoroscopy is of great interest and importance but it must be remembered that for the mechanics of the heart, the motion of the atrio-ventricular septum plays an important rôle; this cannot be visualized under normal circumstances; the proportion between the outer and inner pulsations of the heart may not be identical for all cases.

It is interesting to find out which points along the heart surface are actually causing the x-ray heart silhouette. If one builds a heart model out of clay or plaster of Paris and illuminates it from a point source of light, one imitates x-ray conditions very closely and then one sees that the points forming the heart silhouette are found to be along an irregular line and neither are they all situated in the same plane nor in a plane which is parallel to the frontal plane (our film or screen).

If one uses such a heart model or a real plastic reconstruction of the heart obtained by the method of Palmieri and imitates the x-ray beam by a point source of light, and changes the position of this heart model by minor rotations and inclinations and studies its shadow, one can find: (1) Hearts of different volumes can produce the same shadow area, (2) Hearts of the same volume can produce different shadow areas and a smaller heart can give the same area as compared with a larger one. This is another proof that our aim should be and will be the volumetric determination of the heart size.

III. The left ventricle forms a very small part of the anterior surface of the heart. The bulk of it lies deep. It follows that in order to diagnose commencing hypertrophy, one ought to determine the increase of the ventricular depth. One determines orthodiagraphically the outermost left ventricular contour, shifts the tube with a target film distance of 60 cm., 10 cm. to the right of the patient and determines again the outermost point.

The distance thus obtained is the depth index. If the ventricle increases in depth, the rays meet the border of the heart sooner than in the case of a normal heart and the deflection is greater. Normal figures are from 7 to 14 mm. but again the distance of the left lower cardiac silhouette from the film has great influence.

The normal transverse diameter of the heart may be predicted from the body weight alone in patients of ideal height and weight, but in others, height and weight must be taken into account because the transverse diameter depends greatly upon the build of the individual. The average correction required for height is approximately 1% of the predicted transverse diameter of the heart, for each 2 cm. divergence from ideal height. If for instance, an individual is 10 cm. too short for his weight, 5% has to be added to the predicted transverse diameter. If he is 10 cm. too tall, 5% has to be subtracted. The size of the heart expressed in per cents. of the average normal size for weight and height is useful information but must be considered in relation to the clinical picture as a whole. For example, a heart of plus 10% of predicted normal size in an apparently healthy athlete would not be of much significance but in a sedentary worker with a history of rheumatic fever or with a slightly increased blood pressure, would suggest pathological enlargement.

All of the measurements up to now are mainly based upon studies in the antero-posterior plane, assuming that there is a constant ratio between any diameter obtained in this plane to the depth of the heart. To use an extreme example, is it possible to judge the depth of a house by taking measurements of its front side only?

X-ray studies of 150 healthy people of both sexes and age groups from 15 to 58 years have given interesting results as to the depth of the heart (to be published soon elsewhere). The average depth is about three-quarters of the average long diameter. However, the range of variation is quite marked. In nine-tenths of all cases the depth of the heart may vary from 62.5 to 85% of the transverse diameter and 55 to 75% of the long diameter of the heart. Therefore, we are dealing with different

heart types, shallower and deeper ones. There are hearts which are deeper, smaller and shorter than the average and others which are shallower, larger and longer than the average.

If one studies the chests in which the different heart types are located, one finds an interesting and important relationship: Deeper, smaller and shorter hearts are found in chests which are deeper, smaller and higher than the average; shallower, larger and longer hearts are found in chests which are shallower, wider and shorter than the average.

Thus it is definitely demonstrated that determination of heart size in one plane is insufficient.

IV. Many data have been and still are being collected but the scientific results are proportionately much too small and the words of Pasteur should be recalled: "Nothing can be done without pre-conceived ideas, only there must be wisdom not to accept their deductions beyond what experiments confirm."

Two examples may illustrate the necessity of having experienced men doing x-ray examinations of people applying for insurance. In one case, the patient having been passed, the transverse diameter of the heart was given as 15 cm. and the transverse diameter of the chest 30 cm. If the measurements were correct, then one can say with safety that the heart was diseased; obviously the measurement of the chest was taken not at the inner rib cage but probably from a skin point to a skin point, thus obtaining a "50% ratio". Or the measurement was taken on the fluoroscope without orthodiagraphic measurement, a procedure which is entirely incorrect. Another case was refused by an insurance company on account of "widening of the aorta", the transverse diameter of the vessel shadow being above 7 cm. A study of the film revealed that the patient had a slight degree of scoliosis to the left. The descending aorta was thus displaced to the left. Further studies of the patient under the fluoroscope and films taken so that the curvature of the spine was corrected, revealed an entirely normal size of the aorta.

There are few cardiologists who have sufficient experience to pass judgments as to the x-ray findings of the heart and great vessels and there are few x-ray men who know sufficiently well

the clinical side of the problem. It cannot be hoped that cardiologists will obtain the best possible results when applying the methods of x-ray examinations to their field unless they are taught for a certain amount of time by an experienced man, fundamentals as well as details.

Of course, errors are liable to be made in borderline cases. This is unavoidable, partly because there is a tendency to make definite and positive statements rather than to confess that one is unable to give them. Moreover, one may recall the words of Thomas Huxley: "The only people, scientific or otherwise, who never make mistakes are those who do nothing."

DR. CHRISTIERNIN—The discussion will be continued by Dr. David Steel. Dr. Steel is Rœntgenologist of the Western Reserve University and Lakeside Hospital, Cleveland, Ohio. Dr. Steel is best known for his studies on syphilis of the aorta and for his outstanding work on the relation of the left auricle to mitral disease.

DR. STEEL—Mr. Chairman and Members of the Association: (Presented slides.) An appreciation of the rœntgen method in the study of cardiac disease has been expressed by many of our well known authorities in this country, among whom are R. W. Scott, Paul White, McCræ, Levine, Robey and Christian. It has been my experience that those clinicians who have studied abroad and those clinicians who have been educated in our own clinics where the rœntgenological method is used, have found that it is such a valuable adjunct as not only to warrant its routine use, but also to accept its conclusions in many cases where the rœntgenological and clinical diagnoses differ. I have used the word "adjunct" because I firmly believe that no examination of the heart is complete when only the rœntgen method has been used. Certainly we must agree with Paul White when he says "Rœntgenology is but one of several clinical methods of studying the circulation. * * * A careful history is most important of all in the diagnosis of cardiovascular disease, and physical examination comes next in usefulness. Accessory

and occasional valuable methods of study are sphygmomanometry, rœntgenology and electrocardiography. For the complete examination of a patient with cardiovascular symptoms or signs all these procedures should be included." Even tho we agree in part we must take exception to the statement that it is an occasional or accessory method. In fact I dare say that in many cases the rœntgen method is infinitely more important than either the carefully taken history or the careful physical examination. It is now a well known fact that in larger clinics the rœntgen method far outweighs in importance any other method of examination in *certain* cardiovascular lesions. Suppose we grant that this is only occasionally true in the large clinics of a large metropolitan hospital where expert skill, knowledge and facilities are available, what must the situation be in the rural or suburban districts or in the practitioner's office where of necessity the above advantages of diagnosis are not available and where the very clinical entities susceptible of diagnosis solely by the rœntgen method are only rarely encountered or are mere memories of troublesome lesions in medical school days. I grant that the number of such lesions is not very great, but at the same time I point out that from an insurance point of view these lesions are most important and must form a very large part of your source of error. I deem it a privilege to present my views on some of these lesions before this society and I wish to thank you for the opportunity.

I have enjoyed reading and listening to the paper by Dr. Nichols. For purposes of discussion and to more fully round out the subject, I should like to disagree with his first sentence, namely "The most important single fact in the effort to determine the presence or absence of organic heart disease is the separation of the enlarged heart from one of normal size." I agree that it is important, but I do not agree that it is "the most important". Dr. Nichols has described various methods of measuring this or that portion of the cardiac silhouette and the vascular shadow. He has also discussed the various normal factors which cause an increase in the size of the heart and has sounded a warning that these factors must all be taken into

account when judging cardiac size. It is not always easy to evaluate these normal factors and in addition, the silhouette is not always sharply defined, anatomical relations are many times obscure, normal structures may add to or subtract from the normal silhouette and even with special care it is sometimes difficult to obtain plates in an exact postero-anterior view. Therefore, we often have several factors present, the value of which it is almost impossible to estimate. I believe it is important to realize, therefore, that the various proposed measurements have a decided limitation and that they can often lead to error. Nevertheless, if these limitations are recognized, measurements have a clinical value. Of the various methods, perhaps the simplest and most ingenious is that proposed by Kreuzfuchs; namely, measurement of the aortic width after barium visualization of the aortic bed of the esophagus. This method has been accurate at autopsy in the few cases I have proved. Unfortunately the method measures the aorta near the isthmus rather than near the root where syphilis is more common and more important physiologically. The value of the transverse diameter is so well known that its importance need not be emphasized further than Dr. Nichols has already done. Other measuring methods have value as already conceded, but it seems to me that there are two other methods of studying the pathological heart which are far better than measurements. I refer to the study of the changes in form of the cardiac silhouette and thoro fluoroscopic observation. Just as proficiency grows with experience in other fields of medicine, so does ability to judge slight changes in shape of the cardiac cone increase after many examinations in the normal and pathological. For this reason it seems logical to depend upon experience and general impression after fluoroscopy rather than on a number of figures subject to many unknown factors. To emphasize my point I should like to discuss several lesions which can be properly diagnosed rœntgenologically, yet which do not show an increase in the transverse diameter of the cardiac silhouette and where, if one were to depend on measurements alone, the lesion would be entirely overlooked. I would

also draw your attention to the fact that these lesions to be discussed are usually not diagnosed clinically either because they produce few or no symptoms and signs, or because the signs produced are confusing and not pathognomonic either singly or collectively.

Syphilitic Aortitis

It is stated by Stevens that "about 20 per cent. of all hospital patients and at least 10 per cent. of the entire population give evidence of infection" (syphilitic). Ten per cent. would give us a figure of 12,000,000 cases in this country. Parran states that statistics "warrant the statement that there are in this country 643,000 cases of syphilis constantly under medical care, of which 35 per cent. or 225,000 are early cases and that no less than half a million (500,000) new infections occur for which treatment is sought each year. Statistics would tend to show that 20 to 30 per cent. of those having syphilitic infection develop aortitis. We have the following summary:

Population - - - - - - - - - - - -	122,000,000
Percentage infected with syphilis - - -	12,000,000
Percentage of syphilitics developing aortitis (20 to 30 per cent.)	2,400,000 to 3,600,000

Accepting the yearly crop of new cases of syphilis as 500,000 we would have a ten year crop of 5,000,000. Of these, 20 to 30 per cent. will develop aortitis, giving us a figure of 1,000,000 to 1,500,000 new cases of luetic aortitis in every ten years.

The figures are alarming and are made even more so by the inadequacy of clinical diagnosis. Reid reports that in two large hospitals in Boston the lesion was not diagnosed in 44 per cent. of 78 autopsied cases, in spite of the fact that 34 per cent. of these were considered as advanced. Nor must we lull ourselves into a false sense of security by too great reliance on the Wasserman reaction. The percentage of error might be as high as 40 per cent. It is variously stated as from 15 to 40. In a series of forty proved cases of syphilitic aortitis at City Hospital in Cleveland reported by the speaker, thirty-six were so checked.

Fourteen or 39 per cent. were negative and four, or eleven per cent., were reported as anti-complementary.

We are confronted by a disease which is nearly the commonest and by far one of the most fatal of the serious diseases affecting mankind and yet we must face the fact that over 40 per cent. are correctly diagnosed for the first time at autopsy. If this figure is true of the large metropolitan hospital, what must be the percentage of error in insurance work where examination, to say the least, is by comparison relatively superficial. The error must be high and the cost tremendous.

Aortic syphilis causes no cardiac enlargement unless the valves are involved leading to aortic insufficiency or unless there is a myocardial lesion present. Therefore, all the cases of early luetic aortitis and many of those well advanced could not be detected by cardiac measurements. However, many of these do produce changes in the aortic silhouette and these changes are practically pathognomonic of the lesion. Furthermore, these changes cannot be elicited clinically and hence the rœntgen method is once more in the unique position of being the only means of correct diagnosis. The changes are elicited by both a fluoroscopic examination and by telerœntgenograms. The aortic shadow is seen to be denser than in other lesions, due in part to actual increase in the cross diameter of the blood column incident to dilatation and in part to the changes in the wall. This sign is of supporting value only. The most important sign is irregular dilatation. This presents itself as one or more localized spindle-shaped areas, not aneurysmal in dimension and involving most commonly the root. In the postero-anterior view, such a change appears as a dense shadow of the upper right cardiac arch. Its border if extended would not conform to the remaining portions of the silhouette. In the first oblique the anterior and posterior borders of the ascending aorta do not run parallel as normally, but, because of the greater dilatation of the root, they converge toward the aortic knob. Hence the silhouette is triangular with the base down. If the localized dilatation is near the knob, then the walls converge toward the heart and the silhouette becomes club-shaped. In the second

oblique view, because the course of the aorta is now at right angles to the path of the rays, making the entire silhouette visible especially in the pathological case, local dilatations can easily be made out. These local dilatations are to be expected, and they are easily explained by the disruption of the elastic fibers so common in syphilitic aortitis. With the dilatation there is a localized increase in pulsation which should always be looked for and used as an index to dilatation.

In the later stages, dilatation may become diffuse. This can become extreme, and since marked dilatation is seldom seen in other lesions, it has considerable diagnostic importance. The pulsations are decreased in amplitude, probably due to cicatricial changes. Increasing dilatation on repeated observation tends to rule out atherosclerosis in favor of syphilitic aortitis. A marked degree of generalized dilatation is more common in syphilis and can be taken as presumptive evidence, especially if the heart shadow is small or if it occurs in a patient under forty. When the various segments are variably dilated the diagnosis is almost certain. I say "almost" because Vaquez and Bordet report local dilatation of the aortic root in Hodgkins' disease. Rheumatic dilatation has been reported also.

The fact that slight rœntgen changes are present does not mean that the lesion is pathologically not well marked. In fact I believe that well marked syphilis of the aorta can be present pathologically and yet present no rœntgen signs. The early rœntgen changes consist of some haziness in outline near the root, probably due to infiltration in and about the walls, and localized bulging and increased pulsations which are minimal. It will be noticed that the dilated arch when schematically continued does not conform in outline with the remaining portions of the aorta. Such changes are of even greater significance when found in a patient under forty who gives no history of hypertension and no clinical evidence of organic heart disease. Lues should always be suspected when (1) a diffuse dilatation is present and is associated with a normal sized heart silhouette, (2) when a dense aorta is present in a young individual without a previous hypertension. It can be safely diagnosed when

localized dilatation associated with localized increased pulsation is demonstrable.

Mitral Stenosis

A second and very serious cardiac lesion not associated with enlargement is the not-uncommon case of mitral stenosis which gives either very few and indefinite signs or absolutely no signs except during the brief effect of amyl nitrite inhalations. Such cases can be well advanced and they have necessarily a grave prognosis. A history would put the examiner on his guard, but for obvious reasons in your work a history is not always forthcoming. A mitral stenosis is very easily diagnosed clinically by the murmurs, but if these are absent, the diagnosis will be also. Rœntgenologically a mitral stenosis shows the following changes:

1. The heart is in the erect position and the silhouette appears long and narrow.

2. The left border is straight. The incisura is filled out by the pulmonary artery and left auricular appendage.

3. The left median distance in an uncomplicated case is small.

4. The aortic knob is insignificant or entirely hidden in the shadow of the spine. This is due in part to the habitual scanty filling of the aorta and in part to rotation.

5. The second arch (pulmonary artery) joins the aortic knob high up.

6. The pulmonary artery is increased in prominence due to the rotation.

8. The hilus regions are increased and in about 30 per cent. of the cases the main right descending limb can be measured.

9. The enlarged left atrium encroaches upon the mid-portion of the retrocardiac space and in well marked cases obliterates it, leaving characteristically a clear triangular area of normal radiability in the lower portion.

10. The displaced œsophagus can be demonstrated after ingestion of barium. It will be found to be displaced to the right and riding over the enlarged left auricle.

11. The angle of bifurcation of the trachea will be increased due to pressure of the enlarged left auricle.

The discussion does not apply to mitral insufficiency, for in this lesion the heart is enlarged to the left because the left ventricle undergoes hypertrophy and dilatation as a result of the regurgitation. It hardly seems possible that a well-developed mitral stenosis will escape competent rœntgen observations. The early lesions, when changes in form are minimal, might be quite difficult. In such cases careful fluoroscopic observation of the size and shape of the left auricle offers the only solution. We must not forget that these changes might be very minimal and hence in such cases conclusions drawn from fluoroscopic observation must be guarded.

Coronary Disease

Another serious cardiac lesion often not associated with enlargement is the all-too-common case of coronary disease. Unfortunately we have no rœntgen signs on which a diagnosis can be made, but we do have something to offer which would be of great interest to this society. The amount of calcification of the thoracic aorta is not necessarily an index to similar changes in the coronary arteries. Nevertheless the demonstration of calcium in the aorta by X-ray, the only positive means of identification, certainly means that the individual's life might be abbreviated. Pathologically, extensive aortic calcification is often associated with ulceration and hence thrombosis is always a danger. Such changes are not associated with cardiac enlargement unless myocardial damage has resulted. Demonstration of a pathological aorta should be of interest because of what appears to be a definite increase in incidence of coronary disease. Should coronary thrombosis take place death might follow immediately or after a few days. If the myocardial

infarction undergoes fibrosis and adequate collateral circulation is established, and if the other portions of the myocardium are not decreased enough to impair function, the owner of such a heart will often recover so that his usual duties if not too strenuous, are resumed. His serious handicap then will be elicited only by a careful history, which again might not be forthcoming. I believe that with careful observation more of these cases can be either diagnosed or strongly suspected rœntgenologically. Easy recognition depends upon the presence of bulging beyond the line of the epicardium. If the fibrous scar is not strong enough to resist the intraventricular pressure, this bulging takes place and rœntgenologically shows as a rounded mass which is always continuous with the heart border. Since pathologically the descending ramus of the left coronary is most often involved and since this artery supplies mainly the left ventricle, it follows that cardiac aneurysms are most frequent in the left ventricle. Besides the typical bulging the pulsations are quite characteristic. During systole the intraventricular pressure increases and the aneurysm, unable to withstand this pressure, undergoes dilatation. Therefore we have a systolic expansion with each systole of the ventricle, a condition spoken of as systolic expansion with systolic contraction. When bulging beyond the line of the epicardium is not demonstrable, there may be present only a slight increase in the arc of the upper portion of the left ventricle. Such an increased rounding if associated with abnormal pulsations, especially those pulsations described as systolic expansion with systolic contraction, should make one very suspicious of severe localized myocardial damage. If localized bulging and the above described pulsations are not present, the diagnosis rœntgenologically is not possible. It must also be remembered that if the aneurysm involves an area not susceptible of vision by X-ray, such as the interventricular septum, the diagnosis is also impossible.

Emphysema

Clinically the heart of early emphysema is a small heart. This is also true rœntgenologically. Yet when fluoroscoped it

will show definite evidence of right-sided enlargement. The silhouette will be upright due to rotation brought about by pressure of the enlarged right ventricle against the sternum anteriorly and the diaphragm inferiorly. This rotation is clockwise as it is in mitral disease and the so-called mitral configuration is produced. Rœntgenologically it closely simulates the mitral heart except for one outstanding feature, namely,—the left auricle is not enlarged and hence does not bulge into the retrocardiac space. It is possible to find this chamber not enlarged in mitral disease, but this possibility is so small as to warrant our disregarding it practically. In emphysema the radiability of the lung fields rœntgenologically and the characteristic differences clinically will establish the diagnosis and put the examiner on his guard.

Pericarditis

Adhesive pericarditis may give a silhouette of normal size, yet lead to serious handicaps physically. This has been beautifully shown by Beck of our staff, who has devised surgical relief. The silhouette is often triangular, the pulsations are diminished, respiratory excursion is reduced or absent and changes in position of the heart within the thoracic cage are diminished or absent when the patient is rotated into various postures. If the pericardium is unusually thick all pulsations may be absent and the heart be seriously tamponaded. In advanced cases, clinical signs are present, but it would be very easy to miss the correct diagnosis in a cursory examination. It is not a rare occurrence to find the pericardium the site of an extensive calcification,—the so-called armoured heart. This can be demonstrated solely by X-ray. Should there be localized encapsulated hydropericardium the change in the silhouette might be quite striking, yet not demonstrable by percussion. Typically these lesions have a hexagonal outline. It has been described by Kienbach as "Inflammatory Diverticulum of the Pericardium".

ANEURYSMS

We are all aware of the multiplicity of diagnosis under which aneurysms of the aorta can masquerade, yet I am afraid that not all of us would care to admit that even large aneurysms are quite often present with few or no signs. A well marked aneurysmal dilatation in the sinus of Valsalva cannot be detected by physical examination unless the aortic ring is dilated, producing aortic insuffiency and the dilatation cannot be seen rœntgenologically because this portion of the aorta is hidden from our view. In one case observed a well marked aneurysm of the sinus of Valsalva had ruptured suddenly into the right ventricle while the patient was laboring in a ditch. Yet only a few minutes before he would probably have passed clinically as physically well. I dare say that a careful fluoroscopic examination would have demonstrated his luetic aortitis even tho the aneurysm might not have been seen.

Many examples could be added. I am in hopes that the meagre evidence I have offered is proof positive of

1. The value of routine rœntgen examinations in cardiovascular disease and its value as an insurance routine; and
2. The proof that a rœntgen observation of the heart is not complete when multiple measurements alone are used as an index to a pathological heart, but that
3. The shape of the cardiac silhouette and vascular pedicle is as important or more important than any group of measurements which are subject to so many normal variations, and difficult, if at times not possible, of correlation.

DR. NICHOLS—I have certainly learned a great deal here today. After all, I am a mere tyro sitting at the feet of these masters, and when they disagree with me, it does not hurt my feelings at all. They honor me by coming here and pointing out some of my false impressions.

I am very glad they pointed out and emphasized the value of the early recognition of changes in the contour of the heart. Now that change may be your first clue to heart disease, and

we must learn to recognize incipient changes in contour, because contour changes mean dilatation, which comes before hypertrophy. Long ago a German, Otto Frank, pointed out that the work performed by the heart is a function of the length of the heart muscle fibers. That work was elaborated by Starling and is now recognized by all students of heart muscle physiology. In other words, if you increase the amount of work the ventricle has to do, it can do it only when it dilates or when the muscle fibers lengthen. This means that the very earliest sign of increased heart muscle work is dilatation, thus its contour must change, the permanent changes in form follow later from hypertrophy.

A great many people labor under the misconception that age increases the size of the heart. Dr. Rœsler very definitely showed you today that this is not the case, and I have long believed it. Most of the standards generally accepted for the weight of the normal heart are inaccurate, probably because of the inclusion of cases of hypertension in the later decades. If hypertension is rigidly excluded the heart of the normally aging individual will be a small one.

As far as the position of the septum of the heart in the lateral pictures is concerned, I attempted to do some work on that, using normal fresh hearts at autopsy. As yet I have been unable to satisfy myself that the septum of the heart can be located with sufficient accuracy to warrant its use as an anatomical landmark in applying mensuration to left oblique films. That might work if the septum were a straight line but it is not. It is actually a curvilinear surface and seldom occupies the same position even in normal hearts. Anatomically it's really part of the left ventricle and when the left ventricle enlarges it may be pushed far to the right even to nearly obliterating the cavity of the right ventricle. If you attempt to use it as a landmark, mistakes of one to three centimeters can be expected. I think that this work needs further corroboration.

I owe a great debt of gratitude to these men for coming here and so graciously discussing this paper. They have put pep into what would have been otherwise a very dull communication.

DR. CHRISTIERNIN—May I express to Dr. Rœsler and Dr. Steel the sincere appreciation of the Association for coming here and giving us such able discussions.

The two papers have covered the first two suggestions made by Dr. Birchard in his paper of last year. The third one, which reads in part, is, ". . . . by collaborating with the instrument makers towards the production of apparatus which will give the best possible results." This committee or group has prepared a list of specifications which will be offered to the members of the Association for their assistance in the selection of apparatus. In an adjoining room, there is an exhibition of various types of instruments. Dr. Fellows arranged with various instrument makers to send samples of electrocardiographs, fluoroscopes and X-ray machines, and they are on exhibition in the other room. In the balcony here there is an exhibit which Dr. Ungerleider has prepared of actual insurance applicants who, on physical examination, would have been accepted for insurance had not the special tests been made which brought out conditions which rendered them absolutely uninsurable.

I think it must be evident to you that this committee or group is made up of pretty active men who are thoroughly familiar with the subject in which they are interested. I think it would be a mistake if we allowed this group to separate, and I feel that we should ask the Association to establish them as a permanent committee for our guidance and help, and I would like to have a motion to that effect.

Dr. Willard put Dr. Christiernin's suggestion in the form of a motion, which was seconded and unanimously carried.

AFTERNOON SESSION

DR. CHRISTIERNIN—Before we proceed with the rest of the program, I have asked Dr. Fellows to say a few words concerning the last paper that was read this morning.

DR. FELLOWS—Dr. Christiernin asked me if I would say just one word in connection with the papers which were given this morning. That one word won't be much more than that.

There are a lot of people who do not know technical details and the papers of Dr. Steel and Dr. Rœsler were admittedly technical. They were also scientifically accurate. Dr. Rœsler and Dr. Steel—I have had luncheon with both of them—bear me out in what I say. They are striving and aiming at an ideal. They don't recognize the difficulties with which we have been confronted in issuing life insurance. They don't realize that a fairly good examination of the heart has in some instances been made through an overcoat with an ear. The point is this, that the X-ray examination of a chest and electrocardiographic examination of the heart are unquestionably going to pick out of the applicants looking for insurance the gross abnormality and the case of definite fraud. Now, if we can plug that gap, we have plugged perhaps 75 or 80 per cent. of the leak.

As far as the technical difficulties of arriving at that ideal are concerned, that is what we are up against, and if you were dizzy this morning, you can imagine me sitting with this group of scientists last Spring and again now and probably in the future. So that is what goes on behind the scenes. Actually what goes on in front of the scenes is up on that balcony. Those cases were men who applied for life insurance and would have been accepted only for the fact that a special examination was made and definite abnormalities brought out. There are cases of deliberate fraud. There are cases of men who knew they had nothing wrong. There are cases there who have subsequently developed disability and have gone to hospitals. That is the type of thing that is concrete and definite. That much is

achieved. What we will have in another five or ten years will be better than that and we have got to go through the grief and the worry and the trouble to get those accurate figures.

This is merely because I asked Dr. Christiernin: "Will you tell that crowd not to be discouraged, that it is not quite as much confusion as it sounds?" That's all.

DR. CHRISTIERNIN—The first paper on the afternoon program is entitled, "Blood Pressure Mortality in the Light of Reported Diastolic Readings," and will be presented by Mr. Horace R. Bassford, Assistant Actuary of the Metropolitan Life Insurance Company. Mr. Bassford.

BLOOD PRESSURE MORTALITY IN THE LIGHT REPORTED DIASTOLIC READINGS

By Mr. Horace R. Bassford,

Assistant Actuary of the Metropolitan Life Insurance Company.

Blood Pressure Readings

Any investigation of the effect of blood pressure on mortality should be preceded by a consideration of the reliability of the blood pressure readings which constitute the crude data of any such inquiry.

While the practice of taking systolic pressure dates in the case of a few companies to as early as 1906, it has been recognized that it was not until about 1911 that the taking of systolic pressures became fairly general and not until about 1918 that diastolic pressure was made part of the regular medical examination for life insurance. During this time the technique of taking blood pressure readings underwent gradual improvement. The change from the palpatory to the auscultatory method, the taking of the diastolic pressure at the fifth rather than the fourth phase, and the improvement in instruments can be cited as the purely technical improvements. However, the mastering of the technique of taking blood pressures by the average field physician is perhaps the most important single factor which is likely to be reflected in a statistical investigation of blood pressure readings.

In this latter respect medical examiners undoubtedly became familiar with the taking of the correct systolic pressure at a much earlier date than the diastolic pressure. Even when we admit that on physiological grounds the diastolic pressure is the more fundamental, the more constant and less affected by emotional states, it is still doubtful whether the greater variability of the systolic pressure has not in practice been offset by the greater familiarity of medical examiners with it. It appears not

unlikely that the greater variability of the systolic pressure although accompanied by some corresponding variation in diastolic pressure has been called to our attention largely on account of the ease with which it is observable. On the other hand, the reputed constancy of the diastolic reading is at least partly due to our inability to observe its smaller amplitude of variation. Thus in the case of blood pressure readings in tropical climates, during sleep, and in the case of fever and acute intoxication the fall in diastolic pressure can clearly be observed over a period of time though perhaps not at a given moment.

Statistically, despite the greater variability of the systolic reading, the same absolute error in the taking of systolic pressure results in a much smaller relative error than in the case of the diastolic pressure. Thus a reading within 4 mm. of the true reading in the case of a systolic pressure produces less distortion than the same error in the case of a diastolic reading. When it is considered how pronounced is the tendency to record the terminal digits 0, 8, and how this tendency is aggravated by instruments with two mm. interval scales, the greater range of the systolic pressure may render it easier to decide on the true reading.

In dealing with old diastolic blood pressure material it should constantly be borne in mind that the medical examiners having generally learnt prior to 1918 to take the diastolic pressure at the fourth phase were later asked to change to the fifth phase. In the 1923 Joint Committee Report on Blood Pressure it was indicated that on the issues of 1920 and prior, 29% of the diastolic blood pressure readings were admittedly taken at the fourth phase. It was thought that the fourth phase readings were, because of a gradual change in method, actually fifth phase readings in the more recent material.

On the other hand, the fifth phase diastolic averages were, contrary to expectation, higher than the fourth phase averages. It appears as if a large proportion of the alleged fifth phase readings must have been fourth phase readings. Some confirmation of this theory was given by the blood pressure data furnished by ten companies to the Joint Committee in 1925. The average

blood pressures for 1925 were 5 to 6 mm. under those taken in 1917 or prior and 2 to 3 mm. under those taken 1918-1920. Since the difference between the fourth and fifth phase diastolic may be as much as 6 mm. it is entirely reasonable to suppose that despite the fact that some medical examiners had properly learned to take the diastolic pressure at the fifth phase, the transition from the fourth to the fifth phase was made gradually by the average physician.

The same story is told by the average diastolic readings on Metropolitan policies. The Metropolitan first instructed its medical examiners to take the systolic pressure about 1912. Instructions to take the diastolic at the fifth phase were given in 1917. The following table indicates the average diastolic pressure readings for the periods 1920-21, 1925-26 and 1930 on male white risks accepted by the Metropolitan as standard.

TABLE 1.

Average Diastolic Pressures
Metropolitan White Male Standard Risks

Age Group	1920-1921	1925-1926	1930
20–24	81.6	77.5	75.7
25–29	82.1	78.5	76.6
30–34	83.4	79.5	77.5
35–39	83.3	80.6	78.6
40–44	87.5	81.3	79.3
45–49	86.0	82.3	80.4
50–54	87.5	83.2	81.4
55–59	87.3	84.2	82.3

The most plausible explanation of the above indicated fall in the average diastolic readings was that despite instructions to take the diastolic at the fifth phase, a sizable proportion of physicians continued to take the diastolic pressure at the fourth phase.

Further examination of the 1920-21 diastolic pressure readings led us to believe that in the interest of accuracy the data for these years be best left out. An analysis was made of the

1925-26 readings to determine the tendency to record certain terminal digits and in particular the popular diastolic pressure of 80. The following table indicates the findings of this analysis.

TABLE 2.

Distribution of Diastolic Pressure Readings 1925–26
Metropolitan White Male Standard Risks

	UNITS										
Tens	0	1	2	3	4	5	6	7	8	9	Total
10									1		1
20	1						1			1	3
30	1						1		1		3
40						4	2		5		11
50	29		10		19	9	22		39	2	130
60	322	2	150	7	212	218	184	15	554	24	1688
70	1999	16	581	21	743	800	1014	50	1664	74	6962
80	4462	84	1226	100	1121	1033	874	243	934	181	10258
90	1919	65	353	28	198	188	109	7	91	7	2965
100	118	3	8		3	5	4		3		144
110	13						1				14
120	1		1								2
Total	8865	170	2329	156	2296	2257	2212	315	3292	289	22181
% of Total	40.0	0.8	10.5	0.7	10.3	10.1	10.0	1.4	14.9	1.3	100

The above distribution clearly indicates the popularity of a diastolic reading of 80 and the heaping on the digits 0, 8, 2, 4. This heaping is much the same as was found in the Joint Committee Report in 1925 and is so pronounced as to exercise a decided influence on classification.

The very low (under 60 mm.) and the very high (over 100 mm.) readings when considered in the light of our underwriting rules which provided for their exclusion led us to investigate the possible sources of error in such readings. Only on few of such readings was there a record of other readings taken to verify the correctness of the reading in question. This suggested that aside from possible sources of error from sex, build, racial

and emotional factors which presumably were reported by the physician, the errors in the technic of taking blood pressure might be of particular significance in the case of unusual readings. As against a maximum variability of 20 mm. in the systolic pressure ascribed to prolonged constriction, psychic stimulation or rapid respiration, an unduly prolonged fourth phase has been known to give rise to proportionately lower diastolic readings and occasionally to errors ranging far above the systolic variability.

Consequently on the score of variability and accuracy of blood pressure readings, the existing statistical data do not in our opinion justify very fine subdivisions. In particular the time honored convention of attaching more weight to the systolic reading in old blood pressure material appears to have been based on some sound reasons. Both these points should be borne in mind in the consideration of blood pressure ratings.

A Blood Pressure Experience

An investigation was made of the mortality experience on standard Metropolitan policies issued in 1922, 1923, 1925 and 1926 to white male lives whose systolic and diastolic pressures were recorded. The company rules in 1922 and 1923 provided for the taking of blood pressures on: (a) all Whole Life Policies ($5,000 Min.), (b) other policies of $15,000 and over at ages under 45, (c) other policies of $5,000 and over at ages 45 and over, and (d) otherwise upon Home Office request. When it was found that these groups showed substantially the same extra mortality on account of departures from average blood pressure, the results were combined. In 1925 and 1926 the rules provided for the taking of readings on all policies of $2,500 and over. Only 20% of the issue of these years was included in the investigation, being all the data available without going to the application. The investigation was carried out on the calendar year basis to December 31, 1931, using the Company's Standard Mortality for the same period as the standard of expected deaths. Hence the resulting mortality percentages

reflect without adjustment the variations in mortality on account of various degrees of departure from average systolic and/or diastolic pressures.

Although the diastolic readings in the material under investigation were subject to the same "heaping" errors as were found in the Joint Committee Report 1925 and contained perhaps at the extremes some unreliable readings, it is believed that substantially all the diastolic readings were taken at the fifth phase. Their variation from average as shown in the tables below is based on average diastolic pressures shown on Page 12 of Joint Committee Report—1925. Because of the "heaping" errors and because of the possible variability of the readings the results have been presented in broad subdivisions.

The tables on the following pages set out the experience classified according to departure from average systolic and average diastolic pressures.

An attempt was made to determine whether the absolute diastolic pressure of its departure from average are more significant in measuring extra mortality. The following table shows the mortality experience for all ages combined according to absolute diastolic pressure and the departure from the systolic average:

Metropolitan Life Insurance Company Tables 3, 4 and 5

Mortality Experience Based On Departure From Average Blood Pressures

Issues of 1922, 23, 25 and 26 Carried to Dec. 31, 1931

Ratio of Actual to Expected Mortality According to Company's Standard Mortality for the Same Period

Ages Under 40 at Issue

Millimeters Departure from Average Systolic	Millimeters Departure from Average Diastolic Blood Pressure: -26 to -3	-2 to +2	+3 to +6	+7 to +13	+14 to +21	Total
-25 to -5	84 (241)	97 (57)	81 (12)	51 (5)	— (-)	85 (285)
-4 to +4	100 (262)	84 (261)	107 (178)	99 (164)	92 (8)	95 (873)
+5 to +15	120 (86)	90 (94)	110 (87)	91 (105)	107 (21)	102 (393)
+16 to +30	75 (3)	73 (5)	— (-)	161 (22)	195 (11)	113 (41)
Total	95 (592)	87 (417)	104 (277)	98 (296)	116 (40)	95 (1592)

Ages 40 and Over at Issue

Millimeters Departure from Average Systolic	Millimeters Departure from Average Diastolic Blood Pressure: -26 to -3	-2 to +2	+3 to +6	+7 to +13	+14 to +21	Total
+25 to -5	79 (459)	90 (79)	105 (47)	118 (22)	126 (2)	83 (609)
-4 to +4	107 (628)	103 (272)	98 (258)	96 (188)	121 (31)	102 (1167)
+5 to +15	119 (305)	117 (186)	123 (351)	103 (213)	125 (57)	116 (912)
+16 to +30	151 (21)	171 (17)	184 (40)	214 (54)	119 (19)	174 (151)
Total	95 (113)	106 (554)	111 (696)	107 (477)	123 (99)	103 (1939)

All Ages (15-69) at Issue

Millimeters Departure from Average Systolic	Millimeters Departure from Average Diastolic Blood Pressure: -26 to -3	-2 to +2	+3 to +6	+7 to +13	+14 to +21	Total
-25 to -5	80 (670)	92 (336)	99 (59)	94 (27)	100 (2)	83 (894)
-4 to +4	104 (690)	92 (533)	101 (436)	98 (352)	111 (29)	99 (2040)
+5 to +15	119 (291)	107 (180)	119 (338)	99 (318)	119 (78)	111 (1305)
+16 to +30	133 (24)	131 (22)	144 (40)	196 (76)	139 (35)	157 (197)
Total	96 (1167)	97 (971)	109 (873)	103 (773)	121 (139)	100 (4431)

Figures in parenthesis indicate the number of actual deaths on which the mortality percentage is based.

TABLE 6.

Metropolitan Life Insurance Company Mortality Experience Based on Absolute Diastolic and Departure from Average Systolic Issues of 1922–23–25–26 carried to 12–31–31

Percentages of Actual to Expected Mortality According to Company's Standard Mortality for Same Period

Departure from Systolic Average	ABSOLUTE DIASTOLIC PRESSURE				
	53-74 mm.	75-84 mm.	85-95 mm.	95 mm. & up	Total
—25 to —5mm.	81 (193)	82 (563)	96 (130)	84 (8)	83 (894)
—4 to +4	96 (178)	99 (920)	100 (831)	104 (111)	99 (2040)
+5 to +15	131 (73)	109 (402)	107 (623)	123 (207)	111 (1305)
+16 to +30	135 (6)	111 (28)	175 (100)	160 (58)	156 (192)
Total	93 (450)	95 (1913)	105 (1684)	120 (384)	100 (4431)

The figures in parentheses indicate the actual number of deaths on which the mortality ratios are based.

When the above table is compared with Table 5, it will be seen that an increase in absolute diastolic pressure is equivalent to smaller increments of extra mortality than corresponding departures from average diastolic pressure. This indicates the necessity for taking the age into consideration in determining the effect of diastolic pressure on mortality. This has long been recognized for systolic pressure.

Since the experience cited before is of a relatively short duration, a test was made of the effect of duration on blood pressure mortality. From the experience on the issues of 1920-23 it appeared that the relative mortality of over-average diastolic risks showed a substantial increase over that shown in the early years.

From the preceding it appears that the significant conclusions are as follows:

1. An increase in systolic pressure is more serious than a proportionate increase in diastolic pressure—*for the duration of the experience considered.* When the systolic pressure is normal an increase in diastolic pressure even up to 20 mm. above the

average does not appear to result in a substantial extra mortality. The effects of an increased diastolic pressure probably are not reflected in increased mortality until the longer durations which are not included in this experience.

2. The experience clearly indicates that where both systolic and diastolic pressures are high the resulting mortality is higher than where only one is high.

3. The effect of high blood pressures on mortality is more pronounced at ages over 40 than under 40.

4. A subdivision of the experience according to systolic pressure indicates that although the group of under-average systolic pressures presents the most favorable mortality, very low (16 mm. or more under-average) systolic pressures show increased mortality. The same general statement may be made of under-average diastolic pressures though the trend is less clear. In particular, however, the experience on risks with diastolic pressure 11 mm. or more under-average and systolic 16 mm. or more under-average showed a mortality ratio of 124% of the expected based on 24 deaths.

Blood Pressure Ratings

The systolic and diastolic pressures represent respectively the maximum and minimum pressures in the arterial system. The effective pressure within the arterial system appears to be, therefore, the mean of the systolic and diastolic pressures. It would be reasonable to expect that as far as hypertension is concerned this mean pressure might be used as a measure of the degree of hypertension.

However, such a general index of blood pressure mortality would be defective in that it neglects to take account of the unfavorable variations in pulse pressure and the unfavorable mortality of the combination of very low systolic and very low diastolic pressures.

Practically this index may be modified and turned into a scheme for blood pressure ratings for hypertension only. Departures of either systolic or diastolic pressures below their

averages must in the present state of our knowledge be based on such statistical information as we have.

A modification of this index considers the variation of the systolic when the diastolic is held constant at its normal level, with additions corresponding to a rise in diastolic pressure. For the purpose of ratings, it has been developed in the previous section that departures of the systolic and diastolic pressures from their averages rather than the absolute readings constitute the more sensitive measures of blood pressure mortality. The combination of these ideas resulted in the scheme of ratings shown in the schedule below.

A risk is rated on the basis of its departure from average systolic pressure with additions corresponding to its departure from average diastolic pressure. Because of the inaccuracies and variability of blood pressure readings, it was not thought proper to offer ratings for departures of less than 10 mm. from average. The ratings are based on the Metropolitan experience cited in the previous section, with some allowance for the immaturity of the experience as regards mortality to be experienced on account of over-average diastolic pressure.

The recommended ratings are set out as follows:

RATINGS FOR DEPARTURE FROM AVERAGE SYSTOLIC PRESSURE NORMAL DIASTOLIC PRESSURE

Departure from Average Systolic	Ages at Issue 20–29	30–39	40–49	50–59
10 mm.	0	5	10	10
15 mm.	10	20	25	25
20 mm.	25	30	35	40
25 mm.	35	40	45	55
30 mm.	50	50	60	70

ADDITIONAL RATINGS FOR DEPARTURE FROM AVERAGE DIASTOLIC PRESSURE

Departure from Average Diastolic	All Ages
10	10
15	20
20	35

The above ratings in being based on the Metropolitan experience cited before have not been extended farther than the statistical data warranted. The paucity of data for diastolic pressures in excess of 100 mm. and for systolic pressures over 150 mm. renders arbitrary any ratings derived from this experience for blood pressure above these limits.

Such experience as was obtained on the combination of very low systolic and very low diastolic pressures was insufficient in our opinion to serve as a basis for recommendations for rating such risks.

In the application of the above ratings attention should again be drawn to the deferred nature of blood pressure mortality. This applies particularly to persons with over-average diastolic pressures. Since such pressures may be regarded as signs of more rapid aging, the full effects of which are not reflected in an experience of short duration. In determining the diastolic ratings allowance for this fact was made. The sufficiency of this allowance should, however, be checked up by an actual experience of longer duration. The higher mortality due to over-average systolic pressure manifests itself sooner and it is believed that the basis of the systolic ratings are more nearly accurate on this score, as the mortality ratios are in accord with previous experience of longer duration.

General Conclusions

Our investigation indicates that even as late as 1925 and 1926 the diastolic readings taken by Field physicians were not accurate and points definitely to the need for training in taking more significant fifth phase diastolic readings. Until really accurate readings are obtained there will always be some question as to the correct interpretation of the results of investigations of diastolic pressure.

We believe that further investigations might be made along the lines of studying the simultaneous variations of the systolic and diastolic pressures from their averages as presented in this paper. Our own investigation along these lines suggested a

scheme of rating, first the variation on systolic pressure assuming the diastolic pressure to be normal and secondly rating the departure, if any, of the diastolic from its average.

Dr. Christiernin—A second paper, "On the Interpretation of Blood Pressure", prepared by Drs. MacKenzie and Wells of The Prudential, will be reviewed by Dr. MacKenzie.

ON THE INTERPRETATION OF BLOOD PRESSURE

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1. INTRODUCTION

Seventeen years ago, in his first paper read before this association, one of us emphasized that the systolic pressure was not enough, believing that the fundamental pressure was the diastolic and the proper base from which to estimate wear and tear on heart and arteries. From an experimental point of view the diastolic pressure is the best established and its criteria are in closest agreement. The systolic pressure is too dependent upon the physical characteristics of the measuring instrument, and varies too much from one region of the arterial system to another in different individuals to have adequate significance. It is more dependent on temporary nervous influences, muscular strain, etc., and as a result shows almost twice as much variability as the diastolic. Representing merely the momentary peak pressure, it does not begin to be as important a measure as the diastolic, which indicates the steady load on the arteries and the minimum load which the heart must primarily overcome.

The systolic pressure is in reality compounded of two pressures, (1) the diastolic base, and (2) the pulse pressure, which indicates stroke volume. The simple average of diastolic and systolic is close to the dynamic mean pressure, which expresses the constant load borne by heart and arteries. In routine selection it is not necessary in every case to compute mean and pulse pressures. A glance at systolic and diastolic may be sufficient to classify a case, but for statistical purposes, we think, the systolic should be considered subsidiary, and mortalities should be determined for the two physiological variables, diastolic and pulse pressures. This is the basis of the present report.

After discussing briefly the criteria which have been developed for blood pressure, and its variability, the mortalities of the Prudential issues from 1911 to 1920, inclusive, carried to the end of 1931 are presented. The number of policies included is 66,869, giving an exposure of 632,073.5 years. Analysis shows that the errors in technique by the examiners were not serious, as 99.6 per cent of the systolic readings lay between 100 and 149 mm.Hg. Selection throughout the period was based largely on the systolic. The diastolic readings were apparently not so good, as but 92 per cent lay between 60 and 99 mm. There was difference of opinion amongst the companies as to whether to use the fourth or fifth point. The fourth phase was more than 6 mm. in 7 per cent, approximately (MacKenzie, 1920), and this may have had some bearing. Some may feel that because of inexperience of the examiners the readings of that period were not as reliable as those of today. No tables that we have seen have made us feel that the differences, if any, are important.

The instrumental errors of the epoch are less serious than blood pressure variability. The nervous coordination of the circulation must be faced and interpreted correctly before one can diagnose circulatory fitness. Much more experience is needed along these lines, but we believe we already have good ground for a revision in diastolic and systolic ratings consistent both with medical theory and with mortality experience to date.

2. BLOOD PRESSURE CRITERIA

It is axiomatic that any criterion of measurement should yield a reading independent of the instrument used. No method for blood pressure approaches this ideal. In fact, the very attempt to measure blood pressure changes its value, as has been only too well demonstrated on the rabbit by McGregor (1928) for the systolic. This is explained by the physics of the propagation of waves in elastic tubes, which shows that when blood is not too viscous the pulse bounces back and forth in the arteries, every obstacle reflecting every incident wave and so changing the resultant pressure. To add to the confusion, the muscular

coats of the arteries vary in tone, every nerve stimulus affecting the measurement.

For practical purposes the blood is an incompressible fluid. The velocity of sound in water is over 1,500 meters per second, almost five times its velocity in air, and this measures approximately the rate at which pressure is propagated through the blood. Hence if the arteries were rigid the pulse wave would pass over the entire circulation in a few thousands of a second, practically instantaneously. Actually, it is delayed by the elasticity of the arteries, as will be shown later. If the blood were at rest, the static pressure over the entire system would be the same except for gravity effects. The fact of the matter is that the pulse wave is a volume wave, due to the passage of a certain amount of blood. It is a local piling up of blood, which stretches the artery and produces the pressure.

Bernouilli's energy theorem teaches us that the pressure head is reduced whenever the velocity head, due to the kinetic energy of the moving blood, is increased. Their sum, the total energy, remains nearly constant as the wave passes down the artery. The pressure head in the aorta is measured approximately by the end pressure in arteries branching at right angles from the main artery. If the branch is not at right angles, some of the velocity head is added, and this is why the pressure in the femoral is higher than that in the subclavian, in the horizontal position. Gladstone (1929, 1) has thus explained Dawson's (1906) direct data on the dog completely. When vertical, the femoral pressure is still greater because the gravity head is added.

At present, the broad cuff, 12-15 cm. in width, is universally accepted as standard for adults. In this country the stethoscope is preferred to the oscillometer for both systolic and diastolic. No complete theory of the Korotkow sounds has yet been given, although Gladstone (1929, 2) has outlined their mechanism quite satisfactorily. He explains the silent gap as due to the reduction of pressure head below cuff pressure by the great increase in velocity head as the blood races through the partially constricted brachial artery under the cuff. He, however, fails to emphasize that the velocity head in the brachial must also reduce the ob-

served diastolic below the true diastolic, which is the minimum lateral pressure in the aorta.

There seems to be general agreement that the auscultatory criterion for the systolic is higher than the maximum lateral pressure in the aorta. P. Fabre (1932) has explained this by the well known fact that when a solitary wave is reflected from a ligatured artery the maximum pressure at the ligature is just doubled by the impact of the wave on the dead end. Russell (1908) feared that the resistance of tissues and artery was a serious error, but McWilliam and Kesson (1913) demonstrated that this error seldom amounted to more than a few millimeters, and this has been amply confirmed by other investigators. The auscultatory systolic, therefore, can be accepted usually as about 10, or at most 20 mm., above the true value, when the artery is relaxed.

Vasoconstriction produces a serious increase in the apparent systolic. Indeed McWilliam and Kesson (1913) obtained contraction errors surpassing 100 mm. in excised arteries from the horse and ox at room temperature, and 60-64 mm. errors at body temperature. This shows the importance of checking high systolic records by further observations, and explains some of the extreme variability encountered, particularly in the systolic pressure. The diastolic criterion suffers less from this error, first, because the low pressure in the cuff is less severe as a stimulus, second, because the increase in pressure is partially offset by the lowering due to increased velocity head in the constricted artery. A high diastolic, however, must be interpreted as due to vasocontriction, for Fahr (1932) and his coworkers have shown that arteriosclerosis in itself produces a corresponding fall in diastolic for a given cardiac output.

It is generally recognized by both physiologists and clinicians that the fundamental pressure in determining the work of the heart is not the systolic or the diastolic, but the dynamic mean pressure, the ratio of work done to volume flow. Once the dynamic mean pressure is known, its product by the minute volume gives the load on the heart directly, quite regardless of the heart rate. Now, the minute volume is fixed by the metabolic needs

of the body, which must be met, so that the importance of the dynamic mean pressure is evident. Unfortunately, this pressure cannot be measured directly in clinical work.

Pachon and R. Fabre (1921) have advanced the ingenious theory that the point of maximum oscillations measures the dynamic mean pressure. This has produced a wave of enthusiasm for the oscillometric method in France, Vaquez (1931) even proclaiming a "new era" in blood pressure measurements. He considers this mean to be the true basal pressure and emphasizes its constancy, that it is independent of sex, menstruation, digestive activity, exercise, even of compensated aortic regurgitation. Indeed, he asserts this "oscillometric mean pressure" to be verily a hydraulic constant, analogous to the chemical constant, pH, of the blood. Such exaggeration needs no comment.

Pachon and R. Fabre (1923) admit that for a single pulse the maximum oscillations are at the diastolic, and even when intermittent pulses are observed; but when the pulses are periodic, succeeding each other as frequently as in the circulation of the blood, they maintain that the oscillation maximum gives the true dynamic mean pressure. They present oscillograms from an artificial circulation model to prove it. P. Fabre (1932), however, does not accept their proof, but insists that the oscillations must depend upon such details as lengths, diameters and elastic constants of the mechanical model. He asserts that it is only when these characteristics depart very widely from those of the normal circulation, that any such result is obtained. The sensitivity of the instrument must also enter.

While the cogency of P. Fabre's criticisms must be admitted, in practice the oscillometric mean pressure may yet be useful. If the mechanical characteristics of the oscillometer are standardized, particularly the sensitivity of the vibrating system, and the volume of the cuff is kept fixed (Blavier, 1931), as well as its elasticity, the pressure at which maximum oscillations occur will have an empirical relation to the diastolic pressure, which is all that can be claimed for the auscultatory criterion. It seems probable, therefore, that the oscillograph of Boulitte, which is carefully standardized, sensitive, and uniform in its response,

will be of good service. It has already demonstrated its value in the clinic by revealing local disturbances in the arteries and in studying intermittent claudication (Gőnczy, 1931).

A dynamical study of the mean pressure, to be published later, shows that the true dynamic mean is not so far below the arithmetic mean as has been generally assumed. In fact, if the elasticity of the aorta obeyed Hookes Law the dynamic mean would be considerably above the simple average of diastolic and systolic. When the elastic constants determined by pulse wave velocities are introduced, however, the ratio of work to volume flow is reduced to a value close enough to the simple average of diastolic and systolic so that for practical purposes this average should not lead us astray in clinical interpretation. We believe that its usefulness has not been sufficiently appreciated, and advocate its consideration in medical selection.

These recent researches have clarified the experimental situation. All criteria for the systolic are definitely high, all diastolic criteria definitely low. Probably Marey's classic, the low edge of the maximum oscillations, is nearest the true diastolic, because the two errors involved, (1) the velocity head error, negative, and (2) the oscillometer error, positive, nearly compensate. The change of tone in the stethoscope is somewhat lower than the true diastolic, and the routine criterion (disappearance of sound) still lower. But the simple average of this diastolic and the systolic should roughly approximate the true dynamic mean, which is the important pressure.

Bramwell and Hill (1922) have studied exhaustively the velocity of the pulse wave in man, showing that it depends chiefly on the elastic condition of the arterial wall. Bramwell, Downing and Hill (1923), after extensive measurements, conclude: "Clearly it is impossible for an individual with a high diastolic pressure to attain a considerable output per beat without developing a dangerously high pressure, and throwing an enormous strain on the heart. Conversely, it is obvious that an individual with a low diastolic pressure will be able to reach a high output without difficulty." They present a table which illustrates their results. We give it in Table 1, as these relations must be

understood to appreciate the effect of arterial stiffness upon the heart. It is seen that with a diastolic of 70 and a pulse pressure of 50 the hypothetical stroke volume is 110,

TABLE 1

DIASTOLIC PRESSURE

Pulse	50	70	75	80	100	125	150	175
Pressure	Hypothetical Stroke Volume (cc.)							
25	130	73	64	55	30	24	17	15
50	200	110	97	83	55	41	31	28
75	230	134	122	105	72	56	45	40
100	260	155	139	125	87	70	57	—

whereas when the diastolic has reached 100 with the same pulse pressure the stroke volume is halved. "Quite apart from danger to the arteries, no man with a diastolic between 125 and 150 could possibly take hard exercise for any length of time. He would inevitably suffer from oxygen want."

3. BLOOD PRESSURE VARIABILITY

Very little literature exists on the variability of diastolic and pulse pressure, so we have studied our standard flarimeter tests[1] to see what they show. The changes from Test I to Test IV are perhaps the best standardized measures available, certainly better than the haphazard variations to be found on the medical examiners' reports. For the applicant has concentrated on a standard performance (Tests II and III), so that the vagaries in his pulse and blood pressure are characteristic of him and cannot be ascribed to differences in what he has been asked to do. The median differences between the values in Tests I and IV for first-class risks are Zero, which allows us to assume that in normal risks the individual variability is purely emotional.

A summary of the results on 969 cases with the old technique (with 3 blows in Test III) is presented in Table 2. Table 3 gives a similar summary on 1,080 standard risks (Standard test), and Table 4 shows that of the two series combined (2,049 cases).

[1]See our Progress Report on Flarimeter Tests at last year's Meeting.

TABLE 2

BLOOD PRESSURE VARIABILITY

(Old technique on 969 Adult Males without impairment Except Blood Pressure)

Row		Diastolic (mm.)	Ages (Yrs.)					
			All	Under 20	20-29	30-39	40-49	50 & Over
1	**No. of Cases**	Under 80	645	41	283	194	93	34
2		80 & Over	324	12	87	96	93	36
3	**% of Cases**	80 & Over	33	23	23	33	50	51
			By Percentages					
4	Pulse Pressure	Under 80	38	44	41	35	30	38
5	50 & Over	80 & Over	27	25	23	19	28	61
6	P.P. Variability	Under 80	17	7	18	16	16	29
7	10 mm. & Over	80 & Over	11	17	7	14	11	11
8	Diast. Variability	Under 80	29	17	28	30	37	35
9	5 mm. & Over	80 & Over	16	8	14	10	21	28

TABLE 3

BLOOD PRESSURE VARIABILITY

(Standard technique on 1080 Adult Males without impairment Except Blood Pressure)

Row		Diastolic (mm.)	Ages (Yrs.)					
			All	Under 20	20-29	30-39	40-49	50 & Over
1	**No. of Cases**	Under 80	742	53	316	224	103	46
2		80 & Over	338	5	95	112	84	42
3	**% of Cases**	80 & Over	31	9	23	33	45	48
			By Percentages					
4	Pulse Pressure	Under 80	46	34	51	39	42	57
5	50 & Over	80 & Over	30	20	20	26	29	69
6	P.P. Variability	Under 80	14	19	15	12	9	28
7	10 mm. & Over	80 & Over	14	20	14	12	14	17
8	Diast. Variability	Under 80	32	43	33	30	26	30
9	5 mm. & Over	80 & Over	18	20	17	16	21	17

TABLE 4

BLOOD PRESSURE VARIABILITY

(2,049 Adult Males Without Impairment Except Blood Pressure)

Row		Diastolic (mm.)	Ages (Yrs.)					
			All	Under 20	20-29	30-39	40-49	50 & Over
1	**No. of Cases**	Under 80	1387	94	599	418	196	80
2		80 & Over	662	17	182	208	177	78
3	**% of Cases**	80 & Over	32	15	23	33	47	49
			By Percentages					
4	Pulse Pressure	Under 80	42	38	47	38	36	49
5	50 & Over	80 & Over	29	24	21	23	28	65
6	P.P. Variability	Under 80	16	14	16	14	12	29
7	10 mm. & Over	80 & Over	12	18	10	13	12	14
8	Diast. Variability	Under 80	31	31	31	30	31	32
9	5 mm. & Over	80 & Over	17	12	15	13	21	22

Only adult males were included, those without impairment except for blood pressure. It might be suspected that more cases rated for blood pressure were included at the older ages, but a count of the standard tests (Table 3) gave only 6 over 50, 7 in their forties, 3 in their thirties, 7 in their twenties, and 1 under 20 years of age, so that this age effect of selection must be quite small.

In spite of the small numbers of cases, the frequencies are remarkably systematic, probably because the tests are standardized. The percentage of high diastolics (Row 3) increases steadily with age. There are about twice as many high pulse pressures among the low diastolics (Row 4) as among the high (Row 5). This holds up to age 40 after which the ratio decreases, and for the oldest group there are more high pulse pressures among the high diastolics. Notice that this decrease in the ratio is not due to a decrease in the frequency of high pulse pressures among the low diastolics but to an increase among the high diastolics. It seems obvious that this cannot be a healthy sign. At the younger ages, however, it may be merely a manifestation of the natural nervous lability of youth.

The trend in the variability of the pulse pressure (Rows 6 and 7) confirms this belief, especially in Table 3. The maximum nervous stability seems to be reached at least ten years earlier when the diastolic is high, with a very real increase in pulse pressure variability at the older ages. The variability in the diastolic itself (Rows 8 and 9) shows no consistent trend with age; in fact Row 8, Table 4, is remarkably constant, but it does show at every age much less variability when the diastolic is high. Apparently, as the diastolic increases from childhood to maturity there is a steadying down of the nervous system, both in vasomotor liability (diastolic) and in the regulation of the heart (pulse rate and pulse pressure). Moreover, this stabilization seems to run more parallel with increase in diastolic than with age.

The whole picture is consistent with the conclusion that emotion is responsible for the variability at the younger ages, that the nervous factor stabilizes by the fifth decade of life. More pulse pressure is needed when the diastolic is low, as found, but at the younger ages both heart and arteries can bear more pulse pressure, especially if there is more variability to give periods of relaxation and rest. A lower diastolic must be an advantage if it can mobilize when needed. The increase in variability after age 50, however, is not a good sign, but is perhaps the precursor of established hypertension and later degenerative changes.

There is one possible source of diastolic variability that is worth careful study, namely, changes in the diastolic criterion of measurement. Certain individuals show marked sudden variations in the level of the fourth point and in the length of the fourth phase, from moment to moment. This may be due to vasomotor tone which changes the elastic properties of the arteries and so alters the character of the sounds heard in the stethoscope. This may explain the relative changes in length of the Korotkow phases. For the present, it seems safe to consider diastolic variabilities one-half the systolic variability, or less, as normal, those between one-half and once the systolic variability as showing that the nervous control of the arteries is more labile than that of the heart, while a diastolic more variable than the systolic of the same subject should be considered "atypical".

The diastolic criterion is more difficult than the systolic but insurance statistics have long shown it to be usually less variable in practice.

4. PRUDENTIAL BLOOD PRESSURE MORTALITY

Most mortality experience is available by systolic pressure. It is sufficient to demonstrate that the relative mortality at a given age increases with the systolic above normal. However, there is some doubt that the normal systolic characteristic of health increases with age from 20 to 40 years, although after 40 the systolic normally must increase for physiological reasons. Available statistics indicate that the increase in the systolic averages with age parallels the increasing diastolic, the pulse pressure remaining constant up to age 50 after which it increases more and more rapidly.

About eight years ago a preliminary study was made of Prudential ordinary issues by diastolic and pulse pressure, which showed that the mortality was greatest at the younger ages, at a given diastolic pressure, as briefly summarized in Table 5.

TABLE 5

MORTALITY OF STANDARD RISKS BY DIASTOLIC PRESSURE

Prudential Ordinary Issue 1913-1920, Carried to 1923

Ages at Entry	Diastolic Pressure (mm.) Under 70	70-79	80-89	90 & Over	Under 70	70-79	80-89	90 & Over
	% Actual to Expected (A.M.S.)				Actual Deaths			
15–29	60	77	100	104	8	48	113	78
30–39	47	67	64	81	9	73	160	176
40–49	47	55	63	60	5	42	135	149
50 & Over	26	25	60	62	1	7	49	98
ALL	49	61	69	72	23	170	457	501

The effect of pulse pressure was somewhat ambiguous, as its range was small, but the results indicated not more but less pulse pressure could be allowed the higher the diastolic. This experience has now been carried to the end of 1931. The mortalities by diastolic pressure are summarized in Table 6. All

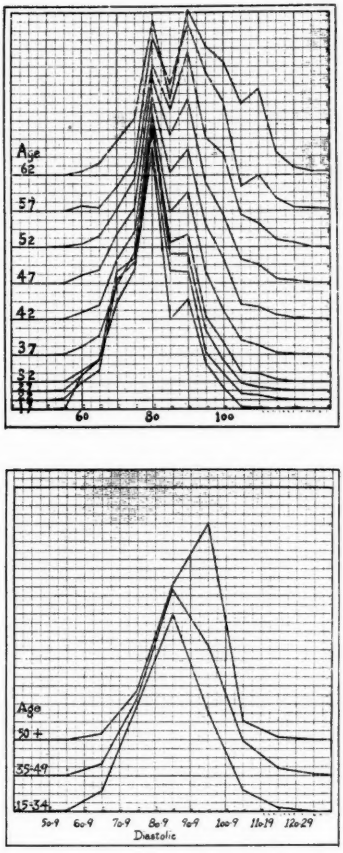
policies issued at standard rates and all those rated because of weight, occupation or family history were included. Expected deaths were computed by the American Men Select Table. To obtain sufficient numbers the risks are grouped by 10 mm. diastolic intervals, but they are given for all ages at entry by 5 mm. grouping in Table 7.

The minimum mortality is consistently at diastolic pressures from 75 to 79 mm., for all insurance years in Table 7, and from 70-79 mm. quite steadily for most age groups and insurance years in Table 6. These pressures represent the optimum as determined in the field under the conditions of life insurance examination in the second decade of this century. It might be suspected that more recent measurements of the fifth point would be appreciably lower. The complete diastolic distributions by age are, therefore, presented in Table 8, and charted in Fig. 1. The median diastolic is 80 mm. up to 35, 85 mm. at ages 35 to 50, and after 50 it is 90 mm. These show clearly the tendency of examiners to report pressures with the last digit either 0 or 5, as has been well demonstrated in the past. The systolics of 10,000 first-class risks accepted early in 1932 showed exactly the same effect. Indeed, in view of the variability of the blood pressure, it is arbitrary to attempt to interpret values closer than 5 mm.

A series of 1,400 flarimeter reports on first-class risks from selected examiners gives median diastolic pressures just 5 mm. lower than those of Table 8 for each age group. We may therefore rest assured that at present the diastolic criterion is not much below what it was fifteen years ago. The systolic and pulse pressures give similar results.

The chart shows the tendency for the peaks of the distribution curves to increase in diastolic slightly at the older ages, but the mode is almost constant up to age 50, as Alvarez and Stanley (1930) found for the systolic. They interpret this to mean that the only effect of age upon blood pressure is to include an increasing proportion of pre-hypertensives, which raises the averages for the older age groups. There is a tendency toward this in our results.

FIG. 1



DISTRIBUTION OF STANDARD RISKS BY DIASTOLIC PRESSURE

TABLE 6

MORTALITY OF STANDARD RISKS BY DIASTOLIC PRESSURE

Prudential Ordinary Issue of 1911—1920 (incl.) Carried to 1931

Diastolic Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
	Insurance Years 1–21													
Under 70	65	65	65	62	66	68	57	91	58	33	21	37	25	8
70–79	63	65	59	67	64	64	46	537	318	219	108	210	172	47
80–89	73	74	72	82	71	74	67	1477	746	731	229	517	533	198
90–99	82	89	78	92	88	79	78	1199	516	683	125	391	439	244
100–109	94	107	88	92	110	94	81	426	148	278	26	122	167	111
110 & Over	106	88	112	88	88	105	117	143	26	117	4	22	51	66
All	76	77	76	80	77	77	74	3873	1812	2061	513	1299	1387	674
	Years 1–5													
Under 70	56	59	51	63	56	59	26	28	20	8	9	11	7	1
70–79	67	74	55	79	72	62	35	193	135	58	52	83	49	9
80–89	70	74	66	96	64	67	63	458	269	189	109	160	139	50
90–99	74	87	64	95	84	63	65	326	175	151	51	124	98	53
100–109	79	107	64	156	93	76	47	100	48	52	16	32	36	16
110 & Over	71	80	67	59	86	65	69	24	7	17	1	6	8	9
All	71	79	63	92	72	65	58	1129	654	475	238	416	337	138

TABLE 6—Continued

MORTALITY OF STANDARD RISKS BY DIASTOLIC PRESSURE

Prudential Ordinary Issue of 1911—1920 (incl.) Carried to 1931

Diastolic Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
	Years 6–10													
Under 70	71	77	63	78	76	69	47	41	27	14	10	17	11	3
70–79	58	58	57	52	61	63	44	203	114	89	32	82	71	18
80–89	68	64	71	59	65	77	59	555	254	301	62	192	229	72
90–99	77	76	78	78	84	71	80	445	185	260	39	146	160	100
100–109	88	94	85	53	113	76	97	151	47	104	5	42	53	51
110 & Over	86	29	102	66	23	107	98	42	3	39	1	2	19	20
All	71	69	73	62	71	73	72	1437	630	807	149	481	543	264
	Years 11–21													
Under 70	68	55	87	31	67	80	102	22	11	11	2	9	7	4
70–79	64	61	66	70	58	70	58	141	69	72	24	45	52	20
80–89	84	89	80	93	88	79	82	464	223	241	58	165	165	76
90–99	98	100	96	107	98	103	86	428	156	272	35	121	181	91
100–109	113	120	110	57	136	127	89	175	53	122	5	48	78	44
110–119	152	160	148	167	159	147	149	70	15	55	2	13	24	31
120 & Over	132	94	142	—	104	—	278	7	1	6	—	1	—	6
All	90	89	91	87	90	92	88	1307	528	779	126	402	507	272

TABLE 7

INCREASE IN MORTALITY WITH DIASTOLIC PRESSURE

(All Ages at Entry)

DIASTOLIC (mm.)	INSURANCE YEARS 1-21	1-5	6-10	11-21	1-21	1-5	6-10	11-21
	ACTUAL DEATHS				% Actual to Expected (A.M.S.)			
Under 70	91	28	41	22	65	56	71	68
70–74	253	91	101	61	73	78	71	71
75–79	284	102	102	80	55	60	49	59
80–84	855	282	313	260	69	70	62	78
85–89	622	176	242	204	80	71	77	93
90–94	757	216	280	261	81	75	75	95
95–99	442	110	165	167	85	72	81	102
100–104	331	80	119	132	95	82	89	112
105–109	95	20	32	43	91	70	83	116
110–114	100	18	27	55	108	77	81	154
115–119	27	3	9	15	100	45	94	139
120 & Over	16	3	6	7	108	76	110	132
ALL	3873	1129	1437	1307	76	71	71	90

Since hypertension is associated with increased mortality, it might be suspected that the general increase in mortality with age is due in part to the increasing proportion of those with high diastolic pressures. There is nothing to controvert this assumption, but it explains only a very small percentage of the whole increase, for the slight decrease in mortality at the older ages, shown in Table 6, at a given diastolic above normal, is a very small fraction of the increase in the mortality table from all causes.

The average age of the risks under 40 is 31.1 years, and of those 40 and over is 45.4 years.

An inspection of Table 6 indicates that the older group has increased less rapidly in mortality than the American Men Table by about ½% per year. Now, for insurance years 11-21, Table

TABLE 8

DISTRIBUTION OF STANDARD RISKS BY DIASTOLIC PRESSURE

Prudential Ordinary Issue of 1911—1920, incl.

Diastolic Pressure (mm.)	AGES AT ENTRY									
	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60&Over
	% of Cases									
40–44	.1									
45										
50										
55	.1	.2	.1		.1			.1		
60	3.7	1.9	1.7	1.2	.9	.8	.9	.3	.6	.4
65	5.4	3.3	3.5	2.5	2.2	1.6	1.5	1.2	.4	1.3
70	15.3	13.4	9.9	9.0	7.7	6.6	5.5	4.2	2.7	3.8
75	16.7	15.3	13.7	12.5	11.4	9.7	8.3	7.6	5.6	6.3
80	28.5	28.0	29.0	29.0	26.7	24.6	22.7	20.2	19.3	17.1
85	10.0	14.3	15.2	15.5	15.9	16.3	16.4	15.2	14.1	8.4
90	12.2	14.2	15.2	16.4	18.1	18.8	20.4	21.6	20.8	18.4
95	4.9	5.2	6.7	7.3	9.2	10.5	11.2	12.1	15.3	14.2
100	2.3	2.8	3.3	4.3	4.9	6.7	7.6	10.3	12.1	12.5
105	.2	.7	.8	1.0	1.6	1.7	2.6	3.6	2.7	5.9
110	.1	.5	.4	.9	1.0	1.4	2.0	2.6	4.0	7.5
115		.1	.1	.2	.2	.5	.5	.8	1.4	2.5
120	.1				.1	.1	.3	.5	.4	.8
125									.3	.4
130 & Over									.2	.4
Total	99.6	99.9	99.6	99.8	100.0	99.3	99.9	100.3	99.9	99.9
Av. Diast.	81.1	82.8	83.8	84.8	85.9	87.2	88.2	89.8	91.6	91.2
Median	80	80	80	80	85	85	85	90	90	90
	No. of Cases									
40–44	1			1			1			
45		1	1					1		
50		2	7	9	5	2				
55	1	11	15	17	18	8	4	3		
60	30	91	181	192	126	83	50	9	4	1
65	44	160	391	393	318	169	83	30	3	3
70	124	652	1095	1410	1131	714	309	104	19	9
75	135	747	1510	1946	1674	1046	470	187	40	15
80	230	1364	3214	4522	3932	2694	1277	494	137	41
85	81	696	1682	2416	2345	1758	926	373	100	20
90	99	692	1685	2564	2677	2029	1147	528	147	44
95	40	254	744	1137	1353	1128	630	296	108	34
100	19	135	369	667	728	729	428	238	86	30
105	2	34	90	148	237	187	144	89	19	14
110	1	24	53	135	141	153	113	63	28	18
115		4	14	30	31	51	30	20	10	6
120	1		7	10	16	16	18	12	3	2
125		1		1	5	2	2		2	1
130 & Over			1	1		1	1	1	2	1
Total	808	4868	11059	15599	14737	10770	5633	2448	708	239
% of Cases	1.2	7.3	16.5	23.3	22.0	16.1	8.4	3.7	1.1	0.4

6 shows an increase in mortality with diastolic above the normal of about 2.5% per mm. Hence the decrease in mortality with age at a given diastolic is just about the amount that it should be to correspond with the increase in the normal diastolic borders with age, that is, about 1 mm. every 5 years.

The increasing significance of blood pressure selection is beautifully shown by Tables 6 and 7. For the first 5 insurance years the trends are not clear, they begin to develop during the years 6-10, but for years 11-21 the trends are unmistakable.

This suggests definitely that medical selection on the basis of diastolic pressure is of increasing importance the longer the duration of the policy.

To show clearly the changes in mortality with the diastolic pressure, the differences obtained by subtracting each mortality in Table 6 from the one above it are given in Table 9, after dividing by the ratios to 100% of the basic mortalities. The latter are the values for all ages at entry and all diastolic pressures for each insurance year group. This corrects each group to the same basis of reference, 100%. The increments are remarkably uniform, considering the small numbers of deaths in some classes. Only three of the classes are large enough for the mortality differences to be actually significant, namely, those in the ALL AGES column at diastolics of 90 and 100, for all insurance years, and at diastolic 90, for insurance years 11-21. The smoothness of the results, however, leaves little doubt as to the reality of their general magnitude.

When the risks are subclassified by pulse pressure, at constant diastolic, as shown in Tables 10-13, incl., the importance of pulse pressure is quite evident. The mortalities are rearranged in Table 14 at constant ages, to bring out the relations. Those in light-faced type indicate classes with less than 10 deaths. When the diastolic is low the best mortality is at high pulse pressures, and vice versa. Thus, the low mortalities run diagonally upward from lower left to upper right, and the lowest mortalities for most age groups are, naturally, at diastolics 70-79, pulse pressures 40-49 mm.

TABLE 9

CORRECTED MORTALITY INCREMENTS (% per 10 mm.)

At Diastolic (mm.)	All	AGES AT ENTRY Under 40	40 & Over	15-29	30-39	40-49	50 & Over
Insurance Years 1—21 (Basic Mortality 76%)							
70	—3	0	—8	7	—3	—5	—14
80	13	12	17	20	9	13	28
90	12	20	8	13	22	7	14
100	16	24	13	0	29	20	4
110	16	—25	32	—5	—29	14	47
Insurance Years 1—5 (Basic Mortality 71%)							
70	15	21	6	23	23	4	13
80	4	0	15	24	—11	7	39
90	6	18	—3	—1	28	—6	3
100	7	28	0	86	13	18	—25
110	—11	—38	4	—137	—10	—15	31
Insurance Years 6—10 (Basic Mortality 71%)							
70	—18	—27	—8	—37	7	—8	—4
80	14	8	20	10	6	20	21
90	13	17	10	27	27	—8	30
100	15	25	10	—35	41	—7	24
110	—3	—92	24	18	—127	44	1
Insurance Years 11—21 (Basic Mortality 90%)							
70	—4	7	—12	43	—10	—11	—49
80	22	31	16	26	33	10	27
90	16	12	18	16	11	27	4
100	17	22	16	—56	42	27	3
110	43	44	42	122	26	22	67

TABLE 10

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 1–21

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
Diastolic Pressures Under 70 mm.														
20–39	81	75	91	59	87	105	—	5	3	2	1	2	2	–
40–49	84	91	73	70	102	60	100	40	27	13	7	20	7	6
50–59	54	50	64	58	44	71	26	39	24	15	11	13	14	1
60 & Over	47	55	40	67	47	59	24	7	4	3	2	2	2	1
All	65	65	65	62	66	68	57	91	58	33	21	37	25	8
70–79 mm.														
20–39	66	70	60	83	63	55	80	70	45	25	17	28	18	7
40–49	59	59	58	57	60	66	32	273	165	108	54	111	95	13
50–59	65	71	59	74	70	65	48	165	93	72	31	62	51	21
60 & Over	87	105	74	167	84	68	83	29	15	14	6	9	8	6
All	63	65	59	67	64	64	46	537	318	219	108	210	172	47

TABLE 10—Continued

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 1–21

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
	80–89 mm.													
20–39	71	73	69	81	70	70	62	440	271	169	98	173	138	31
40–49	70	74	67	81	71	69	62	758	392	366	113	279	275	91
50–59	82	73	86	83	71	90	82	253	76	177	16	60	109	68
60 & Over	92	137	82	167	128	110	61	26	7	19	2	5	11	8
All	73	74	72	82	71	74	67	1477	746	731	229	517	533	198
	90 mm. and Over													
20–39	82	87	77	90	86	80	72	1010	473	537	123	350	369	168
40–49	94	103	91	99	104	90	92	640	196	444	30	166	255	189
50–59	91	105	88	80	109	79	94	110	20	90	2	18	32	58
60 & Over	88	167	82	—	167	83	82	8	1	7	–	1	1	6
All	87	92	83	92	92	84	83	1768	690	1078	155	535	657	421

TABLE 11

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 1–5

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
Diastolic Pressures Under 70 mm.														
20–39	100	143	—	143	143	—	—	2	2	–	1	1	–	–
40–49	67	71	59	68	74	59	59	11	8	3	3	5	2	1
50–59	49	49	49	63	38	59	—	13	9	4	5	4	4	–
60 & Over	41	34	50	—	63	91	—	2	1	1	–	1	1	–
All	56	59	51	63	56	59	26	28	20	8	9	11	7	1
70–79 mm.														
20–39	80	94	60	85	92	33	167	28	21	7	7	14	3	4
40–49	67	70	60	68	72	73	9	105	73	32	26	47	31	1
50–59	60	73	41	92	63	47	27	50	36	14	16	20	11	3
60 & Over	96	98	95	200	56	118	53	10	5	5	3	2	4	1
All	67	74	55	79	72	62	35	193	135	58	52	83	49	9

TABLE 11—Continued

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 1–5

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
	80–89 mm.													
20–39	68	72	60	94	59	58	70	135	94	41	45	49	32	9
40–49	71	75	66	100	65	68	58	248	145	103	57	88	80	23
50–59	74	75	73	74	76	72	74	71	28	43	6	22	26	17
60 & Over	48	105	31	167	77	32	29	4	2	2	1	1	1	1
All	70	74	66	96	64	67	63	458	269	189	109	160	139	50
	90 mm. and Over													
20–39	76	87	65	101	81	72	51	278	160	118	54	106	89	29
40–49	75	93	66	112	89	62	72	148	60	88	13	47	49	39
50–59	70	156	50	111	164	34	61	24	10	14	1	9	4	10
60 & Over	—	—	—	—	—	—	—	—	—	—	—	—	—	—
All	75	90	64	103	86	61	60	450	230	220	68	162	142	78

TABLE 12

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 6–10

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
Diastolic Pressures Under 70 mm.														
20–39	45	71	—	—	125	—	—	1	1	–	–	1	–	–
40–49	93	110	67	105	113	63	74	18	13	5	4	9	3	2
50–59	60	53	71	56	51	76	50	18	10	8	4	6	7	1
60 & Over	85	103	67	182	56	71	63	5	3	2	2	1	1	1
All	73	77	67	78	76	69	63	42	27	15	10	17	11	4
70–79 mm.														
20–39	50	56	42	67	51	38	61	21	14	7	5	9	5	2
40–49	51	43	53	41	53	58	36	98	56	42	15	41	36	6
50–59	67	64	69	56	68	81	44	70	34	36	9	25	28	8
60 & Over	103	179	50	214	167	41	65	14	10	4	3	7	2	2
All	58	58	57	52	61	63	44	203	114	89	32	82	71	18

TABLE 12—Continued

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 6–10

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
	80–89 mm.													
20–39	65	65	64	60	67	67	55	156	92	64	27	65	53	11
40–49	65	61	67	50	67	72	51	285	133	152	26	107	121	31
50–59	77	58	87	97	49	94	76	100	24	76	7	17	49	27
60 & Over	110	200	109	250	188	140	55	13	4	9	1	3	6	3
All	68	64	71	58	65	77	59	554	253	301	61	192	229	72
	90 mm. and Over													
20–39	74	75	73	76	74	72	75	345	151	194	37	114	128	66
40–49	90	105	84	74	111	80	89	245	78	167	8	70	94	73
50–59	80	65	82	—	74	53	102	40	5	35	–	5	9	26
60 & Over	195	500	180	—	500	167	182	8	1	7	–	1	1	6
All	80	83	79	75	85	74	86	638	235	403	45	190	232	171

TABLE 13

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 11–21

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
Diastolic Pressures Under 70 mm.														
20–39	100	—	250	—	—	286	—	2	–	2	–	–	2	–
40–49	93	90	98	—	122	57	188	11	6	5	–	6	2	3
50–59	53	47	75	53	43	81	—	8	5	3	2	3	3	–
60 & Over	—	—	—	—	—	—	—	–	–	–	–	–	–	–
All	64	55	79	31	67	80	102	21	11	10	2	9	7	3
70–79 mm.														
20–39	70	62	80	104	44	94	32	21	10	11	5	5	10	1
40–49	60	56	64	65	53	71	43	70	36	34	13	23	28	6
50–59	71	81	62	70	86	58	68	45	23	22	6	17	12	10
60 & Over	54	—	88	—	—	57	136	5	–	5	–	–	2	3
All	64	62	66	70	58	70	59	141	69	72	24	45	52	20

TABLE 13—Continued

MORTALITY OF STANDARD RISKS BY DIASTOLIC AND PULSE PRESSURES

Prudential Ordinary Issue 1911–1920, carried to 1931

Insurance Years 11–21

Pulse Pressure (mm.)	AGES AT ENTRY													
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)							ACTUAL DEATHS						
80–89 mm.														
20–39	84	88	80	92	86	84	65	149	85	64	26	59	53	11
40–49	79	90	68	100	88	65	79	225	114	111	30	84	74	37
50–59	99	97	100	75	101	102	98	82	24	58	3	21	34	24
60 & Over	113	83	118	—	100	154	95	9	1	8	–	1	4	4
All	84	89	80	95	88	79	82	465	224	241	59	165	165	76
90 mm. and Over														
20–39	97	104	92	92	108	97	83	387	162	225	32	130	152	73
40–49	119	111	121	115	114	127	112	247	58	189	9	49	112	77
50–59	126	102	130	143	95	160	112	46	5	41	1	4	19	22
60 & Over	—	—	—	—	—	—	—	–	–	–	–	–	–	–
All	105	106	105	97	109	110	97	680	225	455	42	183	283	172

TABLE 14

	Pulse Pressure Diastolic	YEARS 1-21				YEARS 11-21				YEARS 6-10				YEARS 1-5			
		20-39	40-49	50-59	60 & Over	20-39	40-49	50-59	60 & Over	20-39	40-49	50-59	60 & Over	20-39	40-49	50-59	60 & Over
		%				%				%				%			
All Ages	Under 70	81	84	54	47	100	93	53	—	45	93	60	85	100	67	49	41
	70-79	66	59	65	87	70	60	71	54	50	51	67	103	80	67	60	96
	80-89	71	70	82	92	84	79	99	113	65	65	77	100	68	71	74	48
	90 & Over	82	94	91	88	97	119	126	—	74	90	80	195	76	75	70	—
Under 40	Under 70	75	91	50	55	—	90	47	—	71	110	53	103	143	71	49	34
	70-79	70	59	71	105	62	56	81	—	56	43	64	179	94	70	73	98
	80-89	73	74	73	137	88	90	97	83	65	61	58	200	72	75	75	105
	90 & Over	87	103	105	167	104	111	102	—	75	105	65	500	87	93	156	—
40 & Over	Under 70	91	73	64	40	250	98	75	—	—	67	71	67	—	59	49	50
	70-79	60	58	59	74	80	64	62	88	42	53	69	50	60	60	41	95
	80-89	69	67	86	82	80	68	100	118	64	67	87	109	60	66	73	31
	90 & Over	77	91	88	82	92	121	130	—	73	84	82	180	65	66	50	—
15-29	Under 70	59	70	58	67	—	—	53	—	—	105	56	182	143	68	63	—
	70-79	83	57	74	167	104	65	70	—	67	41	56	214	85	68	92	200
	80-89	81	81	83	167	92	100	75	—	60	50	97	250	94	100	74	167
	90 & Over	90	99	80	—	92	115	143	—	76	74	—	—	101	112	111	—
30-39	Under 70	87	102	44	47	—	122	43	—	125	113	51	56	143	74	38	63
	70-79	63	60	70	84	44	53	86	—	51	53	68	167	92	72	63	56
	80-89	70	71	71	128	86	88	101	100	67	67	49	188	59	65	76	77
	90 & Over	86	104	109	167	108	114	95	—	74	111	74	500	81	89	164	—
40-49	Under 70	105	60	71	59	286	57	81	—	—	63	76	71	—	59	59	91
	70-79	55	66	65	68	94	71	58	57	38	58	81	41	33	73	47	118
	80-89	70	69	90	110	84	65	102	154	67	72	94	140	58	68	72	32
	90 & Over	80	90	79	83	97	127	160	—	72	80	53	167	72	62	34	—
50 & Over	Under 70	—	100	26	24	—	188	—	—	—	74	50	63	—	59	—	—
	70-79	80	32	48	83	32	43	68	136	61	36	44	65	167	9	27	53
	80-89	62	62	82	61	65	79	98	95	55	51	76	55	70	58	74	29
	90 & Over	72	92	94	82	83	112	112	—	75	89	102	182	51	72	61	—

TABLE 15

MORTALITY OF STANDARD RISKS BY SYSTOLIC PRESSURE

Prudential Ordinary Issue of 1911–1920 (incl.) Carried to 1931

Systolic Pressure (mm.)	AGES AT ENTRY															
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50-59	60 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50-59	60 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)								ACTUAL DEATHS							
Insurance Years 1–21																
Under 110	79	79	79	66	85	80	76	75	45	27	18	8	19	15	2	1
110–119	66	69	61	74	58	62	56	65	501	321	180	121	200	147	30	3
120–129	70	73	66	83	70	69	59	27	1504	880	624	284	596	489	130	5
130–139	83	88	81	82	89	86	71	80	1307	480	827	87	393	561	231	35
140–149	99	122	94	125	121	91	96	104	467	101	366	13	88	164	163	39
150–159	98	85	99	—	103	129	99	77	43	2	41	—	2	11	21	9
160 & Over	157	141	160	—	200	—	244	199	6	1	5	—	1	—	1	4
All	76	78	76	80	77	77	73	80	3873	1812	2061	513	1299	1387	578	96
Years 1–5																
Under 110	78	107	17	101	110	20	—	—	14	13	1	5	8	1	—	—
110–119	70	74	63	83	68	64	56	68	177	125	52	54	71	43	8	1
120–129	68	75	57	96	65	62	43	47	476	323	153	134	189	126	25	2
130–139	76	85	71	88	84	76	61	92	361	161	200	37	124	137	52	11
140–149	74	120	62	203	105	57	68	67	96	32	64	8	24	28	29	7
150–159	35	—	38	—	—	90	37	—	4	—	4	—	—	2	2	—
160 & Over	83	—	102	—	—	—	588	—	1	—	1	—	—	—	1	—
All	71	79	63	92	72	65	57	66	1129	654	475	238	416	337	117	21

TABLE 15—Continued

MORTALITY OF STANDARD RISKS BY SYSTOLIC PRESSURE

Prudential Ordinary Issue of 1911–1920 (incl.) Carried to 1931

Systolic Pressure (mm.)	AGES AT ENTRY															
	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50-59	60 & Over	All Ages	Under 40	40 & Over	15-29	30-39	40-49	50-59	60 & Over
	% ACTUAL TO EXPECTED DEATHS (A.M.S.)								ACTUAL DEATHS							
	Years 6–10															
Under 110	60	62	52	45	71	56	93	—	13	8	5	2	6	4	1	—
110–119	62	64	59	68	58	58	65	45	187	116	71	41	75	56	14	1
120–129	62	62	62	61	63	68	46	15	536	293	243	78	215	201	41	1
130–139	81	84	80	65	88	81	76	80	511	179	332	25	154	218	100	14
140–149	91	102	89	86	104	84	92	97	171	32	139	3	29	62	62	15
150–159	91	112	90	—	139	59	110	78	16	1	15	—	1	2	9	4
160 & Over	203	132	171	—	454	—	—	278	3	1	2	—	1	—	—	2
All	71	69	73	62	71	73	71	77	1437	630	807	149	481	543	227	37
	Years 11–21															
Under 110	103	64	148	35	76	151	104	185	18	6	12	1	5	10	1	1
110–119	65	68	62	69	67	66	44	106	137	80	57	26	54	48	8	1
120–129	84	90	78	96	87	77	86	27	492	264	228	72	192	162	64	2
130–139	93	98	91	98	98	104	72	70	435	140	295	25	115	206	79	10
140–149	130	148	127	67	160	128	122	145	200	37	163	2	35	74	72	17
150–159	155	139	156	—	154	240	131	131	19	1	22	—	1	5	10	5
160 & Over	177	—	206	—	—	—	—	290	1	—	2	—	—	—	—	2
All	90	89	91	87	90	92	87	96	1307	528	779	126	402	507	234	38

The normal decrease in mortality with increasing age, at constant diastolic, is confirmed at constant pulse pressures. The fact that the decrements are not very different from those for all pulse pressures combined indicates that the normal effect of age on pulse pressure is not large. In fact, there is no need to consider age except for the diastolic, which agrees with the physiology of the circulation. High pulse pressure can be tolerated in the young as an indication of emotional lability, and in the old to compensate for the decrease in the vascular bed. At all ages, those with low diastolics must have higher pulse pressures and can tolerate them, while those with high diastolics must have lower pulse pressures to protect the heart, so that the only borders required for pulse pressures are low for low, and high for high diastolics.

In view of these relations it might be suspected that the mortalities by systolic pressure would parallel those by diastolic pressure. Such is, indeed, the case as shown in Table 15. The mortality increments are about the same as in Table 9, from 2 to 4% per mm. increase in pressure. It is this general parallelism between systolic and diastolic which has enabled both clinicians and medical directors to worry along without any closer approach to the physiology of the question, for the systolic has given a crude summation of both diastolic and pulse pressures.

To improve medical selection, however, it is time to avail ourselves of all the information at hand. The late Dr. Symonds (1922) has shown that the variability of the systolic averages 11 mm., that of the diastolic 8 mm., while the variability of the pulse pressure is only 7.4 mm., although he was misled by percentages into exaggerating pulse pressure variability. It is true that pulse pressure varies with the work of the heart, but for a given metabolism it should remain fairly steady. Indeed, vasomotor tone often alters both systolic and diastolic without any change in pulse pressure. We believe that much of the confusion of thought on the subject has arisen from considering the systolic pressure, which is compound, instead of resolving it into its two simple components, diastolic and pulse pressure.

An illustration of the superiority of this basis of study is the

demonstration in Tables 10-14 that the mortality increases at low diastolic and pulse pressures. To be sure, Table 15 also suggests that the optimum systolic is above 110 mm., but the indications are not nearly as definite as in Table 14. For example, the most outstanding increase in Table 15 is for all ages and insurance years 11-21, namely, 103% at systolics under 110 (18 deaths) and 65% at systolics 110-119 (137 deaths). The increase is 38%, but to be significant it should be at least 44%. In Table 10, however, for ages under 40 at pulse pressures 40-49 we find 91% at diastolics under 70 (27 deaths) and 59% at diastolics 70-79 (165 deaths). The increase is 32%, with a border of significance of 27%. These subclasses, therefore, small as they are, are sufficiently contrasted to give a significant increase in mortality at the low diastolic level. Moreover, they have the added advantage that we know the pulse pressures were the same. In Table 15 we do not know what the diastolics were.

The 103% in Table 15 originates in a single class, ages 40-49, which gives 151% with 10 deaths. This does meet the criterion of significance. When subdivided, this class gives 5 deaths at systolics 100-104, and 5 at 105-109, with mortalities 205% and 120%, respectively. Eleven of the 19 subclasses with systolics under 110 with one or more deaths per class have mortalities above 100%, and only 4 are less than 76%, the total mortality for the entire experience. There can be little doubt, therefore, of the reality of the increase in mortality at low systolics, although the argument would be rather weak if based on two classes with 5 deaths in each, when these might be due merely to two cases of multiple policies on single lives.

Table 14, on the other hand, does not depend on any such slender basis for its argument. On the contrary, the evidence for an increased mortality at low diastolic and pulse pressures is abundantly manifest, without any necessity of going back to the detailed subclasses which Table 14 summarizes. A careful study of Table 14 gives convincing evidence of the great value of classification by diastolic and pulse pressures.

Dr. Fisher (1922) found a low mortality, 35% (26 deaths), at systolics under 100 mm., by the same mortality table as used

in our study. This surprising result does not agree with Table 15, but our results do confirm those of Dr. Symonds (1922) which give an increasing mortality for low systolic pressures, particularly at the younger ages.

When the pulse pressures are added to the diastolic, two classes give the same systolic range, 120-138 mm. These are (1) diastolic 70-79, pulse pressure 50-59, and (2) diastolic 80-89, pulse pressure 40-49. Their mortalities are compared in Table 16. They increase quite consistently with increasing diastolic,

TABLE 16

COMPARISON OF MORTALITIES AT SYSTOLICS 120-138

Diastolic Pressure (mm.)	Pulse Pressure (mm.)	All	AGES AT ENTRY					
			Under 40	40 & Over	15-29	30-39	40-49	50 & Over
			Mortalities (% of A.M.S.)					
			Insurance Years 1—21					
70–79	50–59	65	71	59	74	70	65	48
80–89	40–49	70	74	67	81	71	69	62
Increase		5	3	8	7	1	4	14
			Years 1—5					
70–79	50–59	60	73	41	92	63	47	27
80–89	40–49	71	75	66	100	65	68	58
Increase		11	2	25	8	2	21	31
			Years 6—10					
70–79	50–59	67	64	69	56	68	81	44
80–89	40–49	65	61	67	50	67	72	51
Increase		—2	—3	—2	—6	—1	—9	7
			Years 11—21					
70–79	50–59	71	81	62	70	86	58	68
80–89	40–49	79	90	68	100	88	65	79
Increase		8	9	6	30	2	7	11

except for insurance years 6-10, for which they decrease just as consistently. We are unable to explain the latter effect, but the increase for all ages and insurance years is just ½% per mm. increase in diastolic, or 1% per mm. increase in mean pres-

sure. This is only one-fifth of 2.5%, the general increase of mortality with diastolic. It can be explained by the compensating decrease in the pulse pressure, or by the assumption that in the normal range the increase in mortality is very slow. The table shows clearly, however, that even at constant systolic the mortality does increase with diastolic and mean pressure.

5. INTERPRETATION

We have discussed briefly the physics of the circulation, have commented on the values and effects of diastolic, pulse, mean and systolic pressures, the elasticity of the arteries and transmission of pulse waves, also the relationship of velocity head and pressure head to one another, and to arterial walls and pressures, and finally have presented statistical data by diastolic and pulse pressures, also systolic, derived from the Company's issues 1911 to 1920, inclusive. This has been done in an effort to check the correctness of our present methods of selection and to effect improvement where possible.

Before attempting concrete conclusions from the above, we feel that a brief reference to the auscultatory gap is desirable. When present it usually occurs in the second phase of the Korotkow sounds. Its clinical significance, if any, is not known, but it "is of considerable clinical importance because of the error that may be occasioned by disregarding the possibility of its occurrence" (Mudd and White, 1928). They give, from a study of thirty cases, the average upper limit at 177 mm.Hg. and the average lower at 150 mm.Hg. These figures show a spread of 27 mm. "Concerning the mechanism of production of the auscultatory gap, we may say that normally during the second phase the arterial sound is first dulled and then absent, but the dulling and absence are normally obscured by the murmurs" (Gladstone 1929). When the murmur is absent no sound is heard.

"The silent zone is most frequently observed in the second, or murmur, phase; however, it may be found nearer the upper or lower limits of the pulse pressure" (Mudd and White 1928). If this is so then the gap might be even more serious than when occurring in the second phase.

The authors last quoted report thirty cases in which it was found and state that hypertension was present in 28 (93%), and aortic stenosis in 4 (13%).

We call attention to this gap as a very possible cause of failure of examiners to find hypertension in cases in which it has been previously noted and in which, upon further examination, they also find it. Such cases, though perhaps not frequent, are of sufficient importance to make it seem wise when corresponding to bring the possibility of the gap being present to their attention. We feel they should be advised to always raise the pressure sufficiently high to stop oscillation of the needle or Mercury column, or to note by palpation at the wrist the point at which pulsation ceases; also to continue auscultating, during deflation, until the pressure has fallen to at least 100 mm.Hg., or lower, when still heard.

We accept without question the fourth point as indicating the true diastolic in the brachial, but because "In about 10% of cases it is found impossible to decide where point 4 occurs" (Beaumont and Dodds 1931), as it is often more difficult to determine than the fifth point, and because the difference between the two is not greater than 6 mm. in 93% of the cases, we believe that for practical purposes in our Ordinary work the fifth point is sufficient. When very low diastolics are reported these can be checked by asking for another reading giving both the fourth and fifth points.

We have already stated our belief that it "seems safe to consider diastolic variabilities one-half the systolic variability, or less, as normal". The vast majority of standard cases show very little diastolic variability. This pressure alone, however, would give us only slightly better selection than would the systolic. In fact, our data show that it is much the same (Tables 6 and 15). This is undoubtedly because there is a definite parallelism between the two at all except the older age periods. It is not a satisfactory reason, however, for giving the systolic, which is far less stable, the preference, nor for selecting a case by using the pressure which would give the higher rating. Take for instance a systolic of 155 and a diastolic at 102, at age 40—the systolic, we feel, would call for a "B" rating, which averages

60% extra mortality; the diastolic a "C" rating, which averages 120% extra. The sum of these averages would be 180%, which would permit a "D" rating (average 220% extra). The sum of ratings does not by any means cover the damage done to both heart and arteries. An occasional case with very high pressures will live long and well. We know of one, for instance, whose blood pressure taken by a thoroughly competent examiner has for ten years, to our knowledge, been 230 over 120 to 130. He is still alive. It is rare for one to be endowed with such a splendid heart muscle and arterial walls; anything approaching such a strain could be tolerated by but few. The vast majority would soon give way under such a pressure as 155/102. A systolic pressure of 155, age 40, is 30 mm. above the average for that age (Medical Impairments Ratings, page 15). The mortality ratios, M.I.S. page 103, for 25 mm. and more above the average systolic are for ages 30-39 258%, and ages 40-49, 261%. But the M.I.R. gives this group only 70% extra.

One must bear in mind that the systolic and diastolic pressures represent two very different phenomena. The systolic pressure, which is the sum of the diastolic and pulse pressure, is the result of activity of the left ventricle. The diastolic is the result of arteriolar resistance, which originates in quite different parts of the circulatory system. This system is always influenced by the combination of the two, and not by one alone. The ever changing metabolic demands or changes produced by nervous influences are met by vasomotor and cardiac nerves and by the endocrine glands in such away that under normal conditions the various demands or influences are cared for with a minimum of circulatory strain consistent with the requirements. Sudden demands are met by increased heart action and raised pulse pressure, the diastolic remaining the same or falling, the amount of fall being governed by the greatness of the demand for more blood and the extent of the resulting increase in the capillary bed.

The picture changes under abnormal conditions. The variations are too many to discuss in detail but, broadly speaking, when peripheral resistance increases, from whatever cause, there must be, if it continues, a compensatory rise in pulse pressure

if body functions are to be properly maintained. This not only adds to the strain on the arteries but increases the burden of the heart and diminishes its reserve. Sooner or later the reserve will be reduced to the point where there must be an increase in coronary flow to conserve, as far as possible, the functional capacity of the left ventricle. This can be accomplished only through rise in the diastolic pressure, as it is during diastole that the coronaries are filled, the extent of filling depending on the aortic diastolic pressure. Low aortic pressure therefore means insufficient coronary filling and impaired heart function. When the heart is unable to produce a pulse pressure large enough to properly supply the brain, peripheral resistance increases with increase in aortic pressure and cerebral circulation is relatively improved. With the diastolic rising the pulse pressure must also rise, if the left ventricle is capable of increasing its effort. A limit is reached where such compensation is no longer possible and the pulse pressure begins to decrease with fall in systolic and, frequently, further rise in the diastolic. The limit of these compensations is finally reached and the end inevitable. The duration of the whole process depends on the quality of the material that went into the making of the heart and arteries. Variations in the quality largely account for difference in length of life in different individuals with similar impairments.

While increase in diastolic and pulse pressures is serious because of the additional strain imposed on both heart and arteries, the unfavorable effects of low diastolic and low pulse pressures also may be serious. Attention has already been called to the fact that the nutrition and functional ability of the heart are dependent on the height of the aortic diastolic pressure. When this is low coronary circulation is impaired and the heart undernourished. Strain is, of course, absent, but weakness of the muscle develops and its ability to maintain enough pulse pressure to care for metabolic changes usually diminished. Table 14 gives very definite evidence that low diastolic with low pulse pressure give increased mortalities. We feel that when the diastolic is under 60 mm. Hg. it gives ground for question and when under 50 mm. at any age it is positively abnormal. With

such low pressures but nothing to account for them, possible increase in length of the fourth phase (when the pressure is taken at the fifth point) is to be considered, another reading with both the fourth and fifth points given becomes advisable, as the former is a check on the latter. Many persons have low systolic pressures and live long and active lives. Such persons, in our experience, have nearly always had diastolics of over sixty and are members of families also presenting low pressures. When the diastolic is low, large pulse pressures, where there is no evidence of disease, suggest that the heart is being well cared for and fully capable to carry the load of the high pulse pressure without embarrassment. Certainly the low diastolics, with large pulse pressures, give better mortalities than where both were low.

Drs. Rogers and Hunter, Dr. Fisher and the M.I.S. report have all given data to show that where the systolic is over 5 mm. above the average for age, mortality begins to increase and steadily increases as the number of mm. over the average increases. The high limit above the average systolic for age which permitted standard rating has been given as 15 and some have considered it 12 mm. This implies that a young man of say 20 would be impaired if his systolic was over 135 mm., or by some over 132 mm. We are perfectly willing to agree to this if the diastolic was over 90 mm. But suppose it was only 70 or 80 mm., which are perfectly normal for age 20, we are labeling the boy impaired if he has a pulse pressure of 55 or 65 mm., with a perfectly normal diastolic. Our data based on diastolic and pulse pressure do not justify the label. The same reasoning will equally apply to the other age groups, except that as the diastolic advances with age the same wide limits cannot be allowed the pulse pressure. Later on we present tables which we offer as a suggestion for improved selection.

The above seems to us to justify our belief that the diastolic is more important than the systolic and the safest base from which to judge the significance of blood pressure readings. One may live many years with a high systolic when the diastolic remains within normal limits, provided the heart muscle is good.

As soon as the ventricle begins to give under the strain of the large pulse pressure, the diastolic must rise to support it, and the case becomes definitely more serious.

This brings us to the question of mean dynamic pressure which when multiplied by the volume of blood ejected from the ventricle gives the work done by the heart and the constant strain on the arteries. As the volume ejected cannot be determined in our work, we are unable to accurately estimate the cardiac load. Even if we could we would still be without knowledge as to the quality of the heart muscle—the prime factor.

The mean pressures given in the literature are too low because the time average of the pulse tracing, and not the true dynamic mean, was determined. The fact is that the true dynamic mean is very close to the average of the systolic and diastolic pressures. We believe that were it practicable to use the simple average with the diastolic pressure that it would materially aid in selection. This method, however, would involve computation in each case, and is not recommended, except in rare instances, as it would materially delay selection if used as a routine measure. The diastolic and pulse pressures will, we feel, give sufficient information.

Variability of pressures in the same individual is continually coming up to plague us and it is a real and, so far as we know, an unsolved problem as to how best to treat it. Variability at the time of consideration of a case is not sc difficult, for we know how competent the examiner is and have all the associated conditions that go with an examination under our eye. We think that the variability of the individual mean and pulse pressures are of real help in such cases. Table 17 will be of use in this connection. The chief difficulty is where there are records of previous high pressures. What are we to do with an applicant who three years ago had a blood pressure, one reading, of 180 over 120, and was promptly declined? The present readings are within normal limits, but perhaps just at the upper borders. If the former readings were correct, and that we do not know, he was not and it is very doubtful if he now is insurable. Definite hypertension cannot long exist without enlargement of

TABLE 17

PROBABLE NORMAL MEAN PRESSURES

Age (Yrs.)	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60 & Over
MEAN PRESSURE (mm.)										
Low Limit	70	70	75	75	80	80	80	80	80	85
Normal	92	94	95	96	97	98	99	100	102	104
High Limit	115	115	120	120	125	125	125	125	125	120

Mean Pressure is approximately equal to the average of systolic and diastolic, or the diastolic plus half the pulse pressure.

the heart taking place. In the larger cases, therefore, we could well demand a teleroentgenogram and if no hypertrophy was present then we feel it pretty safe to assume that the pressure, if it ever existed, was due to some very temporary and unusual condition and the subsequent pressure probably normal. In the smaller cases where they cannot come to the Home Office for examination, we would have to depend on our examiner's report as to hypertrophy observed at the time of taking additional blood pressure readings. Frequently we are told that high readings are due to nervousness or excitement at the time of examination. If nervous stress will produce marked fluctuations then these, in all probability, occur frequently and with a growing tendency to repetition. The question is, can such a case be considered insurable and, if so, at standard or increased rating? This will depend on a number of factors, the most important being presence or absence of a distinct circulatory strain in the family history, and especially in the parents, the age, occupation, weight and the height of the diastolic pressure. In a young applicant with normal diastolic but increased systolic we believe a good deal of leniency may be shown when all other features of the case are satisfactory, in spite of the fact that some of the cases are pre-hypertensives. After forty years of age such cases are really in the pre-hypertensive stages and cannot as a rule be regarded as standard risks even though some of the readings may be found within normal limits. After fifty we look on marked fluctuation in pressure as of bad prognostic omen.

6. SUGGESTIONS

The Medical Impairment Ratings of the Joint Committee are based on systolic and diastolic pressure, "whichever requires the higher rating". This "either or" method assumes that the two blood pressure impairments do not combine. Our practice has always been to combine the two pressures because the systolic represents, in addition to the diastolic, the pulse pressure which is quite as distinct from the diastolic pressure as is the pulse rate. Taking the diastolic as a single impairment, with the understanding that the pulse pressure is to be combined as a separate impairment, the diastolic limits shown in Table 18 are recommended.

Taking 2% per mm. as the typical rate of increase in mortality with diastolics under 100 mm., and 4% per mm. above 100, our present borders have a factor of safety ranging from 4 to 16, in their rate of increase. Standard rates, however, are allowed for 20 mm. above the normal for each age, or a 40% increase in mortality. In other words, relative to the steady increase shown in Table 9, our borders are too lenient for small increases in the diastolic above normal, but increase too rapidly once the standard border is passed. Mortality statistics are not yet extensive enough to settle this point, so that for the present it seems best to rest the case on medical theory, which favors our suggested limits.

Tables 18 and 19 may seem startling to most members of the Association, but were only fixed upon after much study and discussion. They seem to us in entire agreement with both the physiology and pathology of the circulatory system and to be well supported by the analysis given above. We recognize, of course, that the pulse pressure limits at the younger ages appear very large and give systolics far above what are now looked on as insurable at standard rates. As the size of these pulse pressures, however, is controlled by the height of the diastolic and the age, their acceptance is quite rational.

Take, for instance, a young man twenty years of age. We believe he is acceptable at standard rates with diastolics of

TABLE 18

PROPOSED DIASTOLIC BORDERS

Ages at Issue	STD.		PRUDENTIAL RATING A	B	C	D
	Min.	Max.	MAXIMUM DIASTOLIC ALLOWED			
15–19	50	90	92	94	97	100
20–24	52	92	94	96	99	102
25–29	53	93	95	97	100	103
30–34	54	94	96	98	101	104
35–39	55	95	97	99	102	105
40–44	56	96	98	100	103	106
45–49	57	97	99	102	105	108
50–54	58	98	100	103	106	109
55–59	59	99	101	104	107	110
60 & Over	60	100	102	105	108	112
			MORTALITY EXPECTED (%)			
		100	130	160	220	320

from 50 to 90 mm. Applying the limits given in Table 19 we find that for a diastolic of 50 mm., a pulse pressure of 45 mm. is required which will give a low normal limit for the systolic of 95 mm. and a low mean pressure of 72 mm. The normal mean for this age is 90. A glance at Table 1 will show that he would have a stroke volume of close to 175 cc., which is more than enough to supply his functional needs. At a diastolic of 90, Tables 18 and 19 show that a 55 mm. pulse pressure is allowed. This would permit a systolic of 145 with a mean pressure of 118 mm., an increase of 28 mm. in mean pressure, which though somewhat high could, we believe, be well borne for many years in an otherwise first-class risk. We certainly do not wish to be understood as thinking that such high pulse and mean pressures would be accepted in any but otherwise first-class risks, and then only after further investigation. In giving any table the extremes must be included and their significance in each case left to the judgment of the medical reviewer. Such pressures occur so exceptionally that when suitably selected

the increase in hazard would be very slight. The same method is used at age sixty and over. Here the diastolic limits, Table 18, are 60 to 100 mm., the pulse pressure limits in Table 19, 40 to 50 mm., giving systolic limits of 100 to 150 with mean pressures of 80 to 125. The 7.5 mm. increases in mean pressure over that of the young man at twenty means much, because it is placing a constant increased load of 7.5 mm. on a heart and arteries in which degenerative changes must, with rare exceptions, be definitely established, while at the younger age the arterial walls are fresh and normal, and greater lability of the nervous system expected.

We know well that the high limits suggested for the younger ages are in excess of the usually accepted normal ratings and are positively contraindicated by the M.I.S. study, page 103.

TABLE 19

PROPOSED PULSE PRESSURE BORDERS

(All Ages)

	PULSE PRESSURES ALLOWED				
DIASTOLIC	Rating—Std.	A	B	C	D
50–59	45–70	40–75	35–78	30–80	27–81
60–69	40–70	35–75	30–78	27–80	25–81
70–79	25–65	25–70	25–75	25–78	25–80
80–89	25–60	25–65	25–70	25–75	25–78
90–99	25–55	25–60	25–65	25–70	25–75
100–104	25–50	25–55	25–60	25–65	25–70
105–112	25–45	25–50	25–55	25–60	25–65

At diastolics 70 and over, pulse pressures as low as 25 are allowed.

The association between the Diastolic and Pulse Pressures is such that where ratings are required great care is necessary. The following rules where combinations exist are suggested.

Standard	+A	rate	A
A	+A	"	B
A	+B	"	C
A	+C	"	D
B	+B	"	D

All combinations higher than B+B decline.

Had the diastolics been used in conjunction with the systolics there can be very little doubt that the mortalities shown for the XI, Y1 and X2 groups would have been very different.

The borders proposed in Tables 18 and 19 depart from the M. I. R. in two directions, (1) our rating for a given diastolic is not maximum at the middle ages but increases steadily as the ages decrease, and (2) our ratings are more severe for high pressures, particularly at the younger ages. Table 6 shows conclusively that the mortality at a given diastolic decreases as age advances, and the Blood Pressure Study (1925) detailed Tables 32-35, in spite of their changing base, show more tendency toward a maximum mortality at the younger ages than toward a maximum in the fifth decade. Our reasons for more caution at high diastolic pressures are based on the physiology and pathology of the circulatory system.

In Tables 18 and 19 will be found diastolic and pulse pressure limits not only for standard risks but also for those requiring ratings and the mortality percentages which cover the various ratings so that any one wishing to try our suggestion may easily do it.

Briefly, diastolic pressure increases with age. Pulse pressure decreases with increasing diastolic, but increases with low diastolic and at the younger ages. The pulse pressures, therefore, decrease as increase in mortality is expected.

It would be a great joy to us if, after all the time, thought and labor devoted to this problem, our suggestions were adopted. Such a radical departure from present methods is hardly to be hoped for at once. We are fully convinced that as time goes on our suggestions, or some modification closely approaching them, will be put into practice. Our hope is that that day will soon come, as we are so thoroughly convinced of the theoretical soundness, practicability and safeness of our views.

Cardio-Vascular-Renal Deaths

When the claims are classified by cause of death, the percentages in the cardio-vascular-renal group show a definite tendency to increase not only with age at death but also with the dura-

TABLE 20

CARDIO-VASCULAR-RENAL MORTALITY

Prudential Ordinary Issue of 1911-1920 (incl.) Carried to 1931

DIASTOLIC PRESSURES / PULSE PRESSURES		90 & Over		80–89	
Ages at Death	Policy Years	20–39	40–49	20–39	40–49
		% of C-V-R to Total Deaths			
Under 40	1 – 5	18	36	35	18
	6 & Over	20	41	22	22
40 & over	1 – 5	36	37	22	27
	6 & Over	43	55	33	44
40–49	1 – 5	23	26	26	28
	6 & Over	36	44	34	39
50–59	1 – 5	48	48	11	22
	6 & Over	43	56	30	46
60 & over	1 – 5	67	50	0	43
	6 & Over	54	60	39	54
		Actual C-V-R Deaths			
Under 40	1 – 5	23	14	24	20
	6 & Over	12	9	10	12
40 & over	1 – 5	51	40	13	36
	6 & Over	290	259	86	201
40–49	1 – 5	22	15	12	25
	6 & Over	81	48	44	79
50–59	1 – 5	25	19	1	8
	6 & Over	119	110	29	79
60 & over	1 – 5	4	6	0	3
	6 & Over	90	101	13	43

tion of the policy, especially in classes with diastolic and pulse pressures above normal, where the mortality is high. Seventeen out of the twenty largest age classes in policy years 6 and over have larger percentages of deaths in the C-V-R group than those of the corresponding twenty classes with durations 1 to 5 years.

We have included in the C-V-R group apoplexy, cerebral hemorrhage, pericarditis, endocarditis, organic heart diseases, angina pectoris, diseases of the arteries, embolism, pneumonia and nephritis. The percentages of deaths agree with the M.I.S. standard, namely, 20% at ages under 40, but ours tend to be about 10% higher than 33%, the standard at ages 40 and over. This may be due to the inclusion of angina pectoris, diseases of the arteries, embolism and thrombosis in the "all other" classes of the M.I.S., for they are just about 10% larger than our "all other" classes. Indeed both these nondescript classes are altogether too large. If there are ten classes of deaths, the

residual class should be less than 10%, if twenty classes it should be less than 5%, etc.

A summary of the percentages of C-V-R deaths is presented in Tables 20-22. The two highest diastolic classes (80-89, and 90 and over) are compared by age and by policy year, at pulse pressures 20-39 and 40-49 in Table 20. The percentages increase quite systematically with age, with diastolic, and with pulse pressure. Evidently, the story is told by the large age groups, and so the results for every diastolic and pulse pressure class, and for all detailed insurance years are summarized in Table 21.

TABLE 21

CARDIO-VASCULAR-RENAL MORTALITY

Prudential Ordinary Issue of 1911-1920 (incl.) Carried to 1931

(All Ages at Entry)

PRESSURES		INSURANCE YEARS						
DIASTOLIC	PULSE	1–5	6 & Over	1	2	3–5	6–10	11 & Over
		% of C-V-R to Total Deaths						
90 & Over	20–39	27	42	22	22	28	36	46
	40–49	36	54	40	37	36	51	58
	50–59	37	66	—	25	44	68	74
80–89	20–39	27	31	50	31	26	33	30
	40–49	23	42	14	26	22	39	46
	50–59	25	42	—	11	31	37	48
70–79	20–39	4	29	—	0	4	14	48
	40–49	17	31	0	8	20	30	32
	50–59	28	32	—	21	31	26	42
Under 70	20–39	50	33	—	100	0	0	50
	40–49	27	28	—	0	43	22	36
	50–59	46	35	—	20	50	28	50
		C-V-R Deaths						
90 & Over	20–39	74	302	2	14	58	125	177
	40–49	54	268	2	10	42	124	144
	50–59	9	57	—	2	7	27	30
80–89	20–39	37	96	1	10	26	51	45
	40–49	56	213	1	17	38	110	103
	50–59	18	76	—	2	16	37	39
70–79	20–39	1	12	—	0	1	3	9
	40–49	18	52	0	2	16	30	22
	50–59	14	37	—	3	11	18	19
Under 70	20–39	1	1	—	1	0	0	1
	40–49	3	8	—	0	3	4	4
	50–59	6	9	—	1	4	5	4

TABLE 22

CARDIO-VASCULAR-RENAL MORTALITY BY AGE AT DEATH

(Diastolic Pressures 90 & Over)

INSURANCE YEARS

PULSE PRESSURE	AGES AT DEATH	1–5	6 & Over	1	2	3–5	6–10	11 & Over
		% of C-V-R to Total Deaths						
20–39	Under 40	18	20	33	19	17	22	16
	40 & Over	36	43	0	39	36	38	47
	40–49	23	36	0	12	26	30	44
	50–59	48	43	—	42	50	41	44
	60 & Over	67	54	—	—	67	54	54
40–49	Under 40	36	41	100	43	26	31	67
	40 & Over	37	55	0	31	39	52	58
	40–49	26	44	0	38	25	39	59
	50–59	48	56	0	25	51	58	55
	60 & Over	50	60	0	0	60	60	61
				C-V-R Deaths				
20–39	Under 40	23	12	2	7	14	9	3
	40 & Over	51	290	0	7	44	116	174
	40–49	22	81	0	2	20	40	41
	50–59	25	119	—	5	20	48	71
	60 & Over	4	90	—	—	4	28	62
40–49	Under 40	14	9	2	6	6	5	4
	40 & Over	40	259	0	4	36	119	140
	40–49	15	48	0	3	12	31	17
	50–59	19	110	0	1	18	53	57
	60 & Over	6	101	0	0	6	35	66

There is no doubt about the general trend. The cardio-vascular-renal deaths increase for just those classes which give an increased total mortality. To make sure that this is not the result of increasing average age, the two largest pulse pressure classes at diastolics 90 and over are analyzed by age at death in Table 22. It shows that at diastolics above normal, in addition to the marked increase in the percentage of C-V-R deaths with age at death there is also a definite increase, at constant age, with the duration of the policy. Thus, not only is medical selection by diastolic pressure of increasing importance the longer the duration of the policy, but the increasing mortality is increasingly due to cardio-vascular-renal causes.

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DR. CHRISTIERNIN—Owing to the inability of Dr. Fisher to be present at the meeting, he has asked Dr. Wenstrand, his associate, to come here and he will open the discussion for him.

DR. WENSTRAND—Mr. President, Dr. MacKenzie and Members of the Association: Dr. Fisher regrets very, very much that he is not able to be here. I shall try to fill his shoes as well as possible. That is a very hard thing to do, as you may know, but in presenting what I have to say in discussing this paper, I want to say as an aside that, of course, I hadn't heard

Mr. Bassford's paper until just now and what I do say will apply equally well to what he told you.

In presenting what I have to say in discussing this paper, please bear in mind that Doctor Fisher has gone over with me all the points brought out and that therefore I am expressing his thoughts as well as my own. I mention this particularly as I am substituting for him. The tables submitted of our own experience have been prepared directly under his supervision. Throughout I shall consequently use the plural personal pronouns.

May we be pardoned for prefacing our remarks with a few historical facts as a basis for our assertions and conclusions?

In November, 1906, we began to take palpatory systolic blood pressures in our home office examinations and in August, 1907, examiners in some two score of the larger cities were asked to include such readings in the blanks of all applicants forty years old or over. Within five years we were receiving blood pressure readings from about 85% of our examiners and at all ages. Doctor Fisher read his first paper on this subject before this Association in 1911. The results were obtained by a study of cases which had been declined because of what we then believed to be a high systolic blood pressure, as well as from accepted risks which included some at the older ages with a systolic tension up to 160 mm. Doctor Theodore Janeway in his book, *The Clinical Study of Blood Pressure,* published in 1904, expressed the opinion that 160 mm. was probably the upper normal limit. In 1912 Doctor Fisher stated that very likely 145 mm. was the upper limit of safety and in 1913 first postulated that any systolic blood pressure which was constantly 15 mm. or over above the average for the age would give an increased mortality. This opinion all subsequent studies have proven to be correct. The Transactions of the Association will show the developments of this phase of medical selection and how it has received more and more attention as the years have passed. Doctor MacKenzie was among the early investigators and it is therefore doubly interesting and instructive to study his present contribution, which has been made in collaboration with Doctor Wells.

Auscultatory systolic and diastolic readings had scarcely been heard of when we began, and I do not recall exactly when we started to use this method but my personal examination cards indicate no auscultatory readings prior to 1913. We did not include these additional requirements on our blank until 1917, though some of the examiners recorded them. Even then we retained the palpatory systolic and continued to do so for several years very largely because all our original averages and mortality findings were based upon this standard. We changed our measuring rod after the Joint Blood Pressure Investigation of 1925 had been published and omitted from the blank the palpatory systolic but retained the diastolic at both the fourth and fifth phases. We mention these details to show that our experience until more recently has been limited to systolic blood pressure studies and in general our selection has been along this line. However, we have looked with much doubt upon applicants who have shown unusual diastolic and pulse pressures, especially where the systolic was borderline. It may be safely said that we have very seldom accepted a risk at any age where the diastolic remained constantly over 100 mm. The same is true of the extremely low diastolics.

It should also be stressed that the Northwestern does not accept sub-standard business with the knowledge that it is such, though we recognize that some such cases no doubt creep in occasionally from one cause or another. This statement is made simply to point out that when it comes to the evaluation of risks for ratings, we have no guide-posts with which to make comparisons and therefore some opinions offered may seem shallow unless this is clearly understood. However, medical selection depends upon mortality statistics and their correct interpretation, so we are all pretty much in the same boat.

It would be a waste of time to try to comment on the Introduction or the Blood Pressure Criteria for the review of the subject has been handled in a most masterly and thorough manner. The reference to "a dynamical study of the mean pressure" which is to be published later introduces a thought,

which is also referred to later, namely that the true dynamic mean, being very close to the average of the systolic and diastolic pressures, may by classification of its variations, supply some of the additional information we are seeking. We shall await with much interest the appearance of this study.

The authors speak of Blood Pressure Variability and the relative accuracy of the systolic and the diastolic pressure. Of course it is well recognized that the systolic varies more than the diastolic, due to several factors, but we believe that in most instances it is possible to determine the actual systolic tension by a series of tests taken during the course of a few minutes, especially if the tests be repeated for example two or three times at different times of the day. We have kept track of cases of this kind for many years and the results are shown in Table A.

TABLE A

Risks Accepted with Variable Auscultatory Systolic Blood Pressure, Where the Initial Reading Was 12 mm. or More Above the Average for the Age but Subsequent Readings Were Within the Normal Range.

Issues of 1908 to 1930, Inclusive, Mortality Carried to the Anniversary in 1931. (A.M. Table)

		First Five Years			After Five Years			All Years		
Age at Entry	No.	Exp.	Act.	%	Exp.	Act.	%	Exp.	Act.	%
16–39	465	9.00	16	177.80	21.51	16	74.21	30.51	32	104.90
40 and over	454	22.51	25	111.10	87.21	94	107.80	109.72	119	108.50
TOTAL	919	31.51	41	130.10	108.72	110	101.20	140.23	151	107.70

Some of these applicants were kept under observation soon after the original high systolic reading until it was demonstrated that the actual tension was well within the normal limits. Others were originally declined and later reconsidered after a further study which showed normal readings. The results are not as favorable as our average but we are quite confident that in this group the diastolic pressure was not a factor. We do not try to explain the mortality findings except to emphasize what we

all know that under certain circumstances it seems to be possible to get lower readings in cases which are essentially hypertensive, and perhaps enough of these were included to increase the mortality. Had the diastolics also been high, we should not have encouraged further observation.

In Table 8 it is noted that average diastolic pressures vary from about 80 mm. at ages 15-19 up to 90 mm. at ages 60 and over. In our Table B some comparative averages are shown, which figures were obtained from examinations made only by well known and well qualified examiners. We therefore believe that these averages are as accurate as any which we have published.

TABLE B

AVERAGE BLOOD PRESSURE FROM 20,735 CASES, OCTOBER, 1922, TO OCTOBER, 1923

	PALPATORY	AUSCULTATORY				
			Diastolic		Pulse	
Age	Systolic	Systolic	4th Phase	5th Phase	4th Phase	5th Phase
20	116 mm.	120 mm.	78 mm.	73 mm.	42 mm.	47 mm.
25	117	121	79	74	42	47
30	118	122	80	75	42	47
35	119	123	81	76	42	47
40	120	124	81	76	43	48
45	121	125	82	77	43	48
50	122	126	83	78	43	48
55	124	128	84	79	44	49
60	126	130	85	80	45	50

We agree with Doctor MacKenzie that the fourth phase, that is, the last loud tone, is the correct diastolic pressure, but nowhere in the paper has it been stated whether fourth or fifth phase readings have been used or if there are some of each. Our fourth phase readings vary from 78 mm. at age 20 to 85 mm. at age 60 and the fifth phase diastolics vary similarly from 73 to 80 mm., showing a uniform difference of 5 mm. throughout. Where so much depends upon the diastolic and pulse pressures

as in this study it seems to us that more care should have been used in obtaining and demonstrating uniformity in the scale.

In Tables 6 and 7 it seems to be true that a definite increase in mortality does not occur until diastolics of over 90 or 95 mm. have been reached and, if these figures represent fifth phase readings, it must be assumed that the true diastolics were 95 to 100 mm. The results in the 11-21 years are very interesting but it is hard for us to believe that where the mortality begins to rise sharply when the true diastolic was from 95 to 120 mm. or over, the systolic was not also considerable higher than the average. The pulse pressure on the average, of course, remains fairly constant at all ages.

In Table 10 it is observed that the higher mortalities are found either where the number of actual deaths is small or where systolic pressures, that is the sum of the diastolic and pulse pressures, are decidedly above the normal limits for the age. The same is equally true in Tables 11-12 and 13 to a more marked degree because necessarily as the whole is divided into smaller parts the numbers involved become fewer and consequently less significant. Table 14 to us indicates the same conclusion that where the systolic is high the mortality also is high, while generally speaking, average systolics give average mortalities. The low systolics will be referred to in the next paragraphs.

Table 15 is in line quite generally with what has been shown in other studies of systolic tensions; that the higher the pressure the greater the mortality. However we do feel that, because of the small numbers, too much stress has been placed on the increased mortalities recorded at some of the lower readings.

In this connection we should like to refer to a study made by Mr. E. G. Fassel, Assistant Actuary of the Northwestern, of our 64,574 cards used in the Blood Pressure Investigation of the Joint Committee and published in Vol. XXVII, Part 2, No. 76, October, 1926, under the title, "Blood Pressure Investigation by the Northwestern Mutual Life Insurance Company". Among other results it confirms Doctor Fisher's earlier statement concerning low systolic blood pressures. The most favorable mor-

tality for all ages is found at 10 mm. under the average, namely 39% (A.M.S.) with 59 actual deaths. At 15 mm. under, it is 40% with 50 deaths. On the other hand, and in corroboration of statements made previously, with the systolic tension 10 and 15 mm. over the average, the mortality is respectively 73% and 78%. The general mortality in this study with 962 actual deaths is 52%. In the diastolic and pulse pressure groupings there are no significant differences. Among Mr. Fassel's conclusions this statement is made: "The systolic curve for all ages combined, as corroborated by the two subsidiary curves for age groups, appears to be more distinct than the curves for diastolic and pulse pressure. Also it appears to have the steepest slope of the three. These considerations suggest the possibility that, of the three types of blood pressure, the systolic furnishes the clearest guide in the selection of risks."

Table 16 does not tell the number of actual deaths and therefore our comments may not be fair, but we cannot agree with the authors' conclusion "that even at a constant systolic the mortality does increase with diastolic and mean pressures". It seems to us that the differences between the two classes are too variable and uneven. This may be due to small numbers, which as always tends to unwarranted conclusions.

Before proceeding to our next results, which have a bearing on this assertion, we should like to refer again to Mr. Fassel's paper, because it may be thought that the percentages differ too much. Fassel's figures are considerably lower for two reasons: First, the data were collected from reports of examiners of exceptional accuracy and, second, the 52% mortality excludes the influenza deaths which occurred during the epidemic. With these included, the mortality was 58%. Mr. Fassel points out that the material coming from selected examiners accounts for "a 10 point better experience than on the general business". Neither of these factors obtain in what follows.

We present herewith Tables C and D, which show two groups with a diastolic between 95 and 100 mm. (fourth phase). In C the systolic was within the normal range and in D the systolic was 12-15 mm. above the average for the age. The difference

TABLE C

Issues of 1915 to 1930 Inclusive, Carried to Anniversary in 1931. A. M. Table.

Risks Accepted Where the Diastolic Blood Pressure Was 95 or Over, and the Auscultatory Systolic Was Within the Normal Range.

		First Five Years			After Five Years			All Years		
Age at Entry	No.	Exp.	Act.	%	Exp.	Act.	%	Exp.	Act.	%
16–39	591	11.17	9	80.57	18.84	8	42.46	30.01	17	56.64
40 and over	835	42.89	36	83.94	94.24	79	83.83	137.13	115	83.86
TOTAL	1426	54.06	45	83.24	113.08	87	76.94	167.14	132	78.97

TABLE D

Risks Accepted with Diastolic Blood Pressure of 95 mm. and Over Where the Auscultatory Systolic Was 12 mm.—15 mm. Above the Average for Age.

		First Five Years			After Five Years			All Years		
Age at Entry	No.	Exp.	Act.	%	Exp.	Act.	%	Exp.	Act.	%
16–39	216	4.59	3	65.36	9.60	8	83.33	14.19	11	77.52
40 and over	311	15.14	13	85.86	33.29	34	102.13	48.43	47	97.05
TOTAL	527	19.73	16	81.09	42.89	42	97.93	62.62	58	92.62

in mortality between the two groups, namely C 79% and D 93%, is about what might be expected with a slight definite increase in systolic pressure, indicating that the diastolic of 95 or over did not influence the results either one way or other. It should be noted that in the totals, all years and all ages, the actual deaths are respectively 132 and 58.

Under the heading, Interpretation, the authors make the following statements: "The vast majority of standard cases show very little diastolic variability. This pressure alone, however, would give us only slightly better selection than would the systolic. In fact our data show that it is much the same (Tables 6 and 15). This is undoubtedly because there is a definite parallelism between the two at all except the older age periods. It is not a satisfactory reason, however, for giving the systolic, which is far less stable, the preference, nor for select-

ing a case by using the pressure which would give the higher rating." A little later it is said, "The system is always influenced by the combination of the two, and not by one alone". Isn't this the crux of the problem after all and how is it possible to separate consideration into the component parts when it has been so well established that the higher the systolic the greater the mortality?

DR. MUHLBERG—I am very sorry indeed that I hadn't the opportunity of reading Mr. Bassford's paper before coming to this meeting, and when Dr. MacKenzie asked me to discuss his paper, I warranted that I had no statistical material to submit, but he was willing for me to indulge in glittering generalities, always remembering that that which glitters isn't always gold, and I told him I would be very glad indeed to jot down a few notes. I felt that whatever contribution I would make would be more along physiological lines than based on statistical data.

It is a somewhat astonishing fact that in spite of our recognizing the significance of diastolic pressure, we have known so little about it and we seem to have lacked confidence in the little we know. There appears to be a feeling of uncertainty amongst Medical Directors, not as to its importance, but as to its practical application. Our examiners are less skilled in taking diastolic pressures than they are in recording systolics. In fact, we ourselves are not in harmony in our instructions to them—some companies favoring the cessation of sounds, others the beginning of the fourth phase; and, most disquieting of all, even the physiologists are not so sure which reading is preferable, but they are reasonably sure that neither is as free from probable error as is the systolic estimation.

After all, it is a most complex problem to ascertain the actual pressure of a moving, fluctuating column of liquid. It is easy to determine the pressure of a static liquid, or the velocity of a uniformly moving liquid, but to determine accurately the pressure of a moving liquid with fluctuating pressures, in tubes that vary in diameter through nerve control, is most difficult—at least, without considerable probable error.

But in spite of these physical defects in our apparatus, it is very likely that our readings give us something that roughly parallels the actual pressure, and if the probable error is not too great, this suffices for insurance work, which after all, is predicated on averages.

It is unfortunate that the acceptable diastolics are confined within a smaller range than are the systolics. If you will examine the recommendations of the Joint Committee—and these recommendations very likely reflect the opinions of the majority of Medical Directors—you will note that a systolic of about 130 and a diastolic of about 90 require no extra rating; but an increase of 20 mms. in the systolic carries the same rating as an increase of 10 mms. in the diastolic. Since many of our examiners are evidently less experienced in estimating the diastolic than the systolic, the narrow range adds to our perplexities, especially since there is a difference of about 4 mms. between the fourth phase and the cessation of sounds.

But in spite of all these theoretical objections, the diastolic blood pressure is probably of greater importance than the systolic, not only on account of its greater constancy, but also because a high systolic with a relatively low diastolic, in the absence of aortic lesions or thyrotoxicosis, is less likely to indicate organic changes in the peripheral circulation, whilst a high diastolic, even with a comparatively low systolic, means something radically wrong somewhere.

What is sadly needed to give us full confidence in evaluating diastolic blood pressure, is either greater skill on the part of our examiners, or more imperatively, greater accuracy in our instruments. If they were not so expensive, the recording sphygmomanometer or the oscillometer might solve the question, but cardiologists differ in their opinion of the value of these instruments. Personally, I have not been very much impressed with the reliability of the Recording Sphygmotonometer, particularly with relation to the diastolic pressures. The readings often lack uniformity and are sometimes difficult to interpret. Heart specialists seem content to estimate the systolic or diastolic within 5 mms. of the actual. Such complacence may be war-

ranted from the clinical viewpoint, but 5 mms. variation in diastolic is of paramount importance for insurance selection.

The studies of physiologists are not re-assuring. A Comparative Study of the Criteria of the Human Diastolic Blood Pressure was made by C. E. King and W. E. Garrey and reported in the American Journal of Physiology, Vol. 85, No. 2, June, 1928. They state:

"Simultaneous graphic records were made on smoked paper of pressure within the arm cuff, of oscillations by the Erlanger method, of volume changes of the forearm, and of the amplified sounds of Korotkow.

"All systolic criteria agree sharply, regardless of variations in manipulative procedure. Such is not the case with the diastolic criteria. The speed of inflation of the cuff, and the rate and method of decompression are important factors in determining at what pressure points the characteristic arm volume and accepted sound pressure changes occur. On basis of plethysmographic and auscultatory methods, the slower the decompression the higher will be the diastolic reading. There is less variation in the oscillatory criterion recently adopted by Erlanger.

"The sounds of Korotkow depend, in part, on the degree of fullness of the vascular bed below the compression cuff. Absolute diastolic readings by the auscultatory method are practically impossible. In order to make such readings, even of relative value, both compression and decompression must be carried out rapidly and uniformly."

I have repeatedly in the past confirmed the fact that diastolic blood pressure readings show marked variations, dependent on the speed of decompression, and I was astonished that so little reference was made to this in our literature or warnings given our examiners.

In order to test the variability in readings by our examiners, I asked two of our most skilled examiners to take systolic and diastolic pressure on 41 individuals. I rigged up a stethoscope, so that by means of a Y-tube both examiners could simultaneously hear the auscultatory sounds through double ear-pieces and a single bell. In this experiment the variations on account

of decompression were eliminated and the examiners did not compare criteria either during or after their readings. The beginning of the fourth phase was used for reading the diastolic. While the study was made on only a small number, nevertheless, the results are more re-assuring than King and Garrey would lead us to suppose.

I may state, incidentally, that these are highly skilled examiners. One of them is particularly skilled in lung examinations. I have had the opportunity repeatedly to check up his lung reports with X-ray examinations. The other examiner is perhaps the most skilled man in Auscultatory work that I have encountered, and he ought to be because for three years he was a violinist in a nationally known symphony orchestra.

In 32 instances out of 41 (78%), their systolic readings were identical or within 2 mms. plus or minus. In 27 instances out of 41 (66%), their diastolics were likewise identical. In 7 out of 41 (17%) there was a difference of plus or minus 6 mms. or more in the systolic reading, and the same ratio prevailed for the diastolic. This limited experiment would indicate a greater harmony for systolic readings than for diastolic. But a 17% difference of plus or minus 6 mms. or more systolic and diastolic is a greater divergence in readings than is comfortable for insurance purposes and clearly indicates the need of some more reliable method for determining blood pressures. And it must be remembered that the experiment was conducted by two skilled and experienced examiners.

There are so many interesting facts developed in the paper of Dr. MacKenzie and Dr. Wells that time will not permit me to discuss adequately more than a few isolated instances.

As an example, Table 6 is important in that it shows even a slightly elevated diastolic pressure, 90 to 99 mms. at young ages, 15 to 29, causes an unfavorable mortality, particularly after eleven years' exposure, and that this tendency is even more manifest at slightly older age (30 to 39) and somewhat higher diastolic pressures (100 to 109). It would seem that a high diastolic at the younger ages presages cardiovascular trouble in years to come, and would suggest that persons inheriting a

weak vascular system begin to display some evidence of it rather early in life. The Medico-Actuarial investigation of blood pressure likewise showed a higher mortality at age 25 or less for over-average diastolic blood pressure.

Table 10 is very interesting in indicating that a low diastolic combined with a comparatively high systolic shows a favorable mortality. Where thyrotoxicosis and aortic regurgitation can be excluded, one might expect this on physiological grounds.

The heart receives its blood supply principally during diastole, owing to the compression of the coronary vessels during the contraction of the heart. But it is most probable that the maximum blood-supply to the heart is delivered during the early diastolic or proto-diastolic period, because the same pressure head that closes the aortic valves injects blood into the partially filled coronaries, and therefore, the heart receives its initial blood supply at a pressure that is greater than the mean diastolic—in fact, this pressure closely approximates the systolic. Thus a heart receives an adequate blood supply and at the same time has an easier task in opening the aortic valves, owing to the low diastolic. Such an arrangement ought to be advantageous to the heart muscle.

The corollary expressed by the essayists that at all ages, high diastolics must have low systolics to take the work off the heart, is warranted both by their investigations and by physiological considerations. This is true especially past age 40.

I am inclined to believe that the actuaries may demur, or at least hesitate to accept the statement that mortality experience correctly interpreted demonstrates that medical selection is of increasing importance the longer the duration of the policy. No doubt with more intelligent selection, especially where our newer methods and better interpretation of data come to play, this will become increasingly more evident.

An investigation by my Company into the presence of a moderate number of casts in the urine showed that these risks had a very favorable death-rate for the first ten years of exposure, but a very unfavorable one after ten years. Casts in the urine, like high diastolic pressures at younger ages, forecast

trouble with the circulatory apparatus in the far future. But whether medical selection can distantly protect the companies from such diseases as cancer, diabetes, pernicious anemia, etc., is somewhat debatable.

Table 16 is an interesting one. This table is a study of a systolic range of 120 to 138, with a diastolic that ranges from 70 to 79 in the first group, and a diastolic that ranges from 80 to 89 in the second group. The mortality, except for 6 to 10 years of exposure, is higher for the higher diastolic group. This table is extremely important in confirming some of the essayists' conclusions.

Personally, I should feel more convinced, if the material were subjected to a further analysis. It is possible, where the diastolic is low—namely, 70-79, and the pulse pressure between 50-59, that the systolics cluster more thickly around the lower 50 P.P. range and that the higher diastolic group may have systolics that are more numerous at the 49 P.P. range. This might make the higher diastolic have an average systolic of almost 5 mms. higher than the lower diastolics, and a difference of 5 mms. in the systolic alone could account for the slight differences in the mortalities of these two groups.

The proposed diastolic borders, Table 18, seem to be a distinct improvement over the recommendations under Code 344 of the Joint Committee report in the Medical Impairment Ratings. It wisely divorces the diastolic ratings from the systolics and increases the ratings for the higher diastolics at the younger ages. Whether the more liberal ratings for diastolics at the older age is warranted, I do not know. It may take some courage to accept, even with a high substandard rating, a risk whose diastolic pressure is reported by a reliable examiner to be 106 mms. or over.

Finally, the essayists are to be congratulated in submitting a paper of greatest importance and interest and one that shows the most painstaking research and profound study.

Dr. MacKenzie—Mr. President and Gentlemen: I appreciate greatly the courtesy of Drs. Wenstrand, Fisher and Muhlberg in

paying our paper the compliment of discussing it. I regret immensely that Dr. Hunter of the New York Life was unable to discuss it. He promised me that he would, but later developments so occupied his time that he said he didn't feel that he was really in a position to do it, and it was only last Monday morning, to my great regret, that I heard from him that he couldn't take part. I am very sorry.

We are very appreciative of the splendid discussion of our paper by Dr. Muhlberg and the support he has given us throughout.

In discussing Table 10 the doctor does not seem quite convinced with our data and suggests that the higher diastolics might have an average systolic of almost 5 millimeters higher.

We admit there is probably some bias toward the lower pulse pressure in each group, but we differ with the doctor about their clustering around a 49 pulse pressure range. An examination of the actual deaths in Tables 10 to 13 indicates that they cluster more thickly around the *40* than the 49 pulse pressure range. This does not seem to vitiate these tables, however, as we are confident that the groups differ in systolic by closer to *1* than to 5 millimeters.

I was glad to note that Mr. Bassford's analysis of his blood pressure and the analysis of our blood pressure correspond almost entirely. He and I were talking about it a few minutes before. I am sorry I did not have an opportunity to see his paper so that I might have studied it a little bit before attempting to say anything. As I didn't have that opportunity, my attempts will be few indeed in that respect.

There is one feature that I do notice. He spoke about the probable incorrectness of the earlier readings. The mode that Mr. Bassford gives for his analysis and our mode are exactly at 80. I think perhaps you noticed that, Mr. Bassford. There may be a few millimeters difference between the readings of 1912 to 1920 and the present readings, but, as we stated in our paper, those differences were not sufficiently marked to in any way vitiate or, at least, to vitiate to any material extent the analysis that we have made of our earlier readings. We took

these readings because of the very considerable exposure that we had. It extended over a period of approximately twenty years. We began to take diastolic readings in 1912. In 1915 I presented a paper to this Association on the diastolic pressure and gave some account there of our diastolic pressures as we had obtained them. They were not coming in as fast as we wished they would, but we were getting some and I reported them.

Now, I am in entire accord with the point—and I believe it is a most important point—that Mr. Bassford brought out, that high diastolics and high systolics spell trouble. We can't get away from that any more than we can get away from the fact that low diastolics with low pulse pressures also spell trouble. One is because, as I said in the introductory remarks, the strain on the heart is so great that it wears it out, and the other is because the heart and brain and body are so insufficiently nourished on account of the very low mean pressure and the low systolic that the mortality is also increased. We asked Dr. Fisher to discuss this paper and I can't tell you how sorry I am that the doctor isn't here. It was very gracious of him to take the trouble to write, through Dr. Wenstrand, any comment on our paper. I am sure you will all agree with me that there is no man in this country in the medical profession who did more to introduce blood pressure and its significance to this country and has done more valuable work on it in the last twenty to twenty-five years than has Dr. Fisher. Therefore, I exceedingly regret that he isn't here. But I appreciate very much Dr. Wenstrand's paper. I am sorry that I did not have an opportunity to see it. It is really so full of meat that it will be a hazardous thing for me, in the few minutes at my disposal and with the brief acquaintance I have with it, to attempt to in any way, if I thought it necessary, refute any of his statements.

We did not in our paper wish to minimize the importance of the systolic pressure. We realize that the systolic and the diastolic have a certain degree of parallelism and we get practically the same mortality from the systolic pressure that we do from the diastolic pressure, taken in large groups, but, Gentlemen,

we are going to get caught in some cases if we depend on the systolic. You can have a very unfavorable diastolic pressure because it is high and a perfectly normal systolic, and those are the cases we are trying to cut out. That can't occur if you use the diastolic and the pulse pressure. That is why we feel that it is much safer to make the basis the diastolic and, as we said before, check by the pulse pressure rather than depend on the systolic pressure, which may look perfectly normal but be absolutely abnormal. I think any clinician will concur in that point of view.

Now, in regard to the fourth and fifth phase, I think most of our readings have been taken at the fifth phase. For two or three years we did ask to have the readings taken at both phases, but we found out, and Dr. Jaquith in his paper—I forget the exact date of it for the moment—read before this Association showed that 93 per cent. of the cases had a fourth phase of not over 6 millimeters, and I know from a study that I made of those cases that the majority of them were 4 millimeters or less. I was very glad to note that Dr. Muhlberg referred to the difference between the fourth and fifth point as about 4 millimeters.

Now, Gentlemen, five millimeters is quite within the probable error. I venture to say that if half a dozen men in this room took the blood pressure on an individual at intervals of five minutes, they would be very apt to get readings that varied more than five millimeters, at least in the systolic, and possibly in the diastolic. So we can not place too much emphasis on some variation in the blood pressure readings. However, I do agree with Dr. Muhlberg absolutely that a change of 5 millimeters in the diastolic pressure is of great significance, of far more significance even than the change of 10 millimeters in the systolic, and that seems perfectly rational when one considers the nerve supply of the vessels, the nerve supply of the heart, and the greater stability of the diastolic.

I don't know that I can add anything to what has been said. I don't want to take up your time with further discussion, but I may say our mortality records, as we have presented them

to this Association today, bear out very fully the conclusions we arrived at some years ago. There are three things we want to emphasize particularly in connection with this paper: First, that high diastolic with a high systolic pressure spells trouble; that a low diastolic with a low systolic spells trouble; that you can have a high diastolic with a normal systolic pressure and be thoroughly deceived in accepting a case unless you take the diastolic with it.

MR. BASSFORD—I just want to make a few remarks. I want to issue a warning that if you are going to compare the results of my paper with those of Dr. MacKenzie, you must realize that the mortality table used in my paper is the Company's current experience, while in Dr. MacKenzie's he used the American Men Select.

In the latter part of his paper he converted that into current experience, but even there if you are going to compare it with the results in my paper, it ought to be compared age by age and not in total.

Our company experience represents about 75% of the American Men Select, so that if on the average the mortality shows 100% of the American Men Select, it means about 133% of the mortality according to the Metropolitan's current table.

With regard to fourth and fifth phase, I am not competent to argue which is the better. My own little thought is that if you are going to get statistics they ought to be based on either one or the other and not on a combination of the two. If a difference of 5 millimeters is important, then it is important to know whether it is a fifth phase or fourth phase reading that you are dealing with.

There is just one further matter, about reading on systolics only. I think my paper shows definitely that if the diastolic is normal, an increase in systolic is not as serious as if the diastolic is above average, and therefore it is necessary to take into account not only the systolic but also the diastolic.

In conclusion, I just want to say that I am indebted both to Dr. Christiernin and Mr. Lew in my office in the prepara-

tion of this paper and I want to thank you for the opportunity of speaking here, even though I have had conferred upon me a degree when I first came here. I was called "Doctor" several times and after I made my talk, every one called me "Mister"—they took my degree away. (Laughter.)

DR. MACKENZIE—Gentlemen, I would just like to say that our readings are based on the fifth and not on the fourth phase, so that one would have to consider the diastolic pressure as at least 4 millimeters higher if they are going to compare it with readings based on the fourth phase.

DR. CHRISTIERNIN—The next paper, "The Law of Small Chances in Medical Selection," will be presented by Dr. Wells.

THE LAW OF SMALL CHANCES IN MEDICAL SELECTION

By P. V. Wells

Prudential Insurance Company of America, Newark, N. J.

Life insurance as a whole is built on Bernouilli's Law of Large Numbers, but medical selection should be guided by Poisson's Law of Small Chances. Just as pure science is the basis of industrial practice, so modern medical theory should lead the way in accepting new classes of risks. As soon as a few deaths occur in such classes, their mortality is compared with expectation, the statistical standard of which is Poisson's Law. It is only systematic departure from this law which can be considered significant.

As the samples increase in size, experience replaces theory, until selection is finally on the sure footing of fact. Such an actuarial millenium, however, is ever being postponed by the rapid advances in medicine. Applicants will not remain ignorant but take advantage of the new information to select against the companies. Progressive medical departments, therefore, would not rely on the mere magnitude of their exposure to risk. Instead, they should study in detail by small samples the changes in selected classes and so keep abreast of the new conditions.

Poisson derived his law as an approximation valid only when the chances are small. The risk of death, in short exposures, usually *is* small. We shall first explain the law and its limitations. To convince the skeptical (as it is wise to be toward statistics) we shall show that the basic data of the M.A.M.I. do obey the law, even when taken in small samples. The errors inherent in mortality measurements are such that for medical studies most information can be obtained at least cost by sampling exposures. Finally, a practical illustration will be given

of the experimental method of attacking selection problems by small samples.

1. A Significance Test

It is a human tendency to be unduly impressed by a closer agreement between mortalities than their probable errors would lead one to expect, only to be disappointed later by the opposite freak of sampling. Apparently, the most surprising fact with which we can be confronted is the experimental verification of the logical rule that every single sample is equally a freak. It is only because so many of the freaks are similar enough to be classed as alike that we call such typical freaks more probable than the rarer varieties. The discipline of the significance test tends to stabilize our attitude toward the fluctuations of sampling. It warns us not to be too optimistic when we are having a run of luck, and it helps us to be conservative when confronted with apparent significance.

The most valuable use of the theory, however, is not to teach conservatism, but efficiency. The main purpose of this paper is to convince you that there are unmined nuggets in small samples. To be sure, the larger the sample the better. But to confuse the issue by lumping together indiscriminately all sorts of risks just to get large samples, is not the solution of selection problems. If contrary influences are combined, they tend to neutralize and the net result may be quite insignificant. Study the risks in sufficient detail, which is the natural instinct of one trained in medicine, and there is some hope of revealing the significant causes of death. Of course, this looks like a counsel of perfection. Have we sufficient detail? And if we had, would not the classes be so numerous that the deaths in each class would be too few to give reliable results? These discouragements may be fatal in some problems, but the significance test should be our guide. Whenever we strike a sub-class with significant mortality, it is worthy of attention no matter how few the deaths.

When the expected mortality before the experience was 100%, but afterwards the actual deaths are taken as the best estimate of the true number, the borders of significance can be computed

once for all and stated directly, as in Table 1. To use the table, simply compare the observed mortality with the border corresponding to the actual deaths. If it is above the upper border or below the lower, it is of such rare occurrence in random sam-

TABLE I.

Significant Mortalities

(When the Mortality Expected was 100%)

Actual Deaths	Above %	Below %	Actual Deaths	Above %	Below %	Actual Deaths	Above %	Below %
0	..	..	30	159	73	100	125	83
1	..	..	31	158	73	110	123	84
2	..	..	32	156	74	120	122	85
3	..	..	33	155	74	130	121	85
4	..	..	34	153	74	140	120	86
5	..	..	35	152	74	150	119	86
6	..	..	36	151	75	160	119	86
7	1316	52	37	150	75	170	118	87
8	610	54	38	149	75	180	117	87
9	433	57	39	148	76	190	117	87
10	351	58	40	147	76	200	116	88
11	305	60	41	146	76	210	116	88
12	274	61	42	145	76	220	115	88
13	252	62	43	144	77	230	115	88
14	236	63	44	143	77	240	115	89
15	224	64	45	143	77	250	114	89
16	214	65	46	142	77	260	114	89
17	206	66	47	141	77	270	114	89
18	199	67	48	141	78	280	113	89
19	193	67	49	140	78	290	113	90
20	188	68	50	140	78	300	113	90
21	183	69	55	137	79	350	112	90
22	180	69	60	135	79	400	111	91
23	176	70	65	133	80	450	110	91
24	173	70	70	131	81	500	110	92
25	170	71	75	130	81	600	109	93
26	168	71	80	129	82	700	108	93
27	165	72	85	128	82	800	107	94
28	163	72	90	127	83	900	107	94
29	161	72	95	126	83	1000	107	94

pling that some other interpretation is highly probable. For instance, suppose we have observed a mortality of 150% with 30 deaths. The table shows that 159% is the minimum mortality above 100% corresponding to 30 deaths, so that 150% is not quite significant. If the number of deaths had been 40, the border 147% shows that 150% would have been significant. Below 100%, the lower border is used in similar fashion but in the reverse direction.

The borders in Table 1 indicate a limit to subdividing classes, namely, that it should never be carried to such a point that subclasses contain less than 7 deaths. The table gives directly the number of deaths required to render a given mortality significant. Thus, 18 deaths are required for 200%, 37 deaths for 150%, 100 deaths for 125%, etc. It is evident how seldom experience is extensive enough to justify stating mortalities closer than 1%, which is significant only for more than 39,000 deaths. On the other hand, a subclass containing 10 deaths with a mortality over 350% is significant, if 100% is expected. This shows the virtue in Dr. Rogers' motto: "If the class is too small, subdivide!"[1]

A frequent problem is the significance of a difference between two observed mortalities which are nearly alike, say two experiences from similar classes of risks. For example, suppose these are 90% (25 deaths) and 120% (100 deaths). Is it probable that such a difference could arise just from random sampling? Now there is a rule in the theory of errors, which also follows quite simply from Poisson's Law, that when the samples are independent the error of a difference is equal to the square root of the sum of the squares of the errors of the samples. It is equivalent to enter Table 2 with a number less than either number of deaths, which can be found by dividing their product by their sum. In our example this is 2500/125=20. In Table 2, 20 deaths gives 47%, and the weighted average mortality is $(25 \times 90 + 100 \times 120)/125 = 114\%$, so that $47 \times 1.14 = 54\%$, which is much larger than the difference 120—90=30%. The

1. See discussion of Dr. Dwight's classic paper on "The Value of Small Classes", Proc. Ass'n. Life Ins. Med. Dir., 1913, pp. 211-231.

probable error of the difference is about one third of 54%, or 18%. The difference, therefore, is $30 \pm 18\%$, nearly twice its probable error, which is somewhat large, but it can hardly be classed as significant, for a difference of at least this magnitude would arise about once in every four random samples.

Table 2 is also useful when the expected mortality is not 100%. Simply multiply the percentage in the table by the ratio of the expected mortality to 100%, to get the significant extra

TABLE II.

Significant Mortality Differences

(When the Weighted Average Mortality is 100%)

Actual Deaths	%	Actual Deaths	%	Actual Deaths	%	Actual Deaths	%
0	..	20	47	40	32	200	14.0
1	..	21	46	45	30	210	13.7
2	898	22	44	50	28	220	13.4
3	248	23	43	55	27	230	13.1
4	159	24	42	60	26	240	12.8
5	124	25	41	65	25	250	12.5
6	105	26	40	70	24	260	12.3
7	93	27	40	75	23	270	12.0
8	83	28	39	80	22	280	11.8
9	77	29	38	90	21	290	11.6
10	72	30	37	100	20	300	11.4
11	67	31	37	110	19	350	10.7
12	64	32	36	120	18	400	9.9
13	60	33	35	130	17	350	9.3
14	58	34	35	140	17	500	8.8
15	55	35	34	150	16	600	8.0
16	53	36	34	160	16	700	7.4
17	52	37	33	170	15	800	6.9
18	50	38	33	180	15	900	6.5
19	48	39	32	190	14	1000	6.2

Mortality Difference $= M_1 - M_2$.

Weighted Average Mortality $= (d_1M_1 + d_2M_2)/(d_1 + d_2)$.

Equivalent "Actual Deaths" $= d_1d_2/(d_1 + d_2)$.

Where M_1 and M_2 are the two mortalities based on actual deaths, d_1 and d_2, respectively.

mortality. Thus, if 50% is expected, 100 actual deaths gives 10% extra, or 60%, as significant. While it is not necessary to be exact in such calculations, this does not mean that the problem is trivial. One should keep constantly in mind a rough idea of the borders of Table 1, for the number of deaths at hand. After some practice one can tell at a glance, from this number, whether the mortality is significant or not.

The significance test will be illustrated by practical examples later in the paper. Before doing so, however, it may be of interest to give a glimpse of the theoretical basis of Tables 1 and 2. Of course, no one need worry about such questions. They can be taken for granted, just as patients take prescriptions. The art of using a tool is quite distinct from that of its fabrication, although the more we appreciate of each other's efforts the happier the solution will be. There is no need for confusion of thought in facing statistical theory. Its technicalities, like those of other fields, are sometimes redundant and unnecessary, which adds to the labor of learning them, but few of the problems are as difficult or as complicated as the correct diagnosis of many a case. Some may desire to skip the next two sections and turn at once to the more practical sections 4 and 5.

2. Poisson's Law.

The theory of probability has had a singularly checkered career, great mathematicians have mistaken its interpretation, and some aspects have been highly developed to the neglect of others just as important. Poisson's theorem is a striking example. First published in 1837, it has taken a century to be appreciated. One of the few groups who make full use of it is here in this city in the Bell Telephone Laboratory. Their mathematician, Dr. Thornton C. Fry, has demonstrated that the law is exact in a population of any size or risk when the deaths occur both individually and collectively at random in time.[2]

Each risk, therefore, must die independently of all the others, so that epidemics, group deaths by accident, etc., may vitiate the

2. See his book on "Probability and its Engineering Uses", Van Nostrand, New York, 1928, pp. 220-240.

ideal conditions somewhat if they cause an appreciable proportion of deaths. Naturally, the randomness requires that there be no trend in the death rate with time, but this is not serious if the exposures are short. A more practical difficulty is the correlation introduced when taking exposures by policy. Small samples should always be studied by lives.

Now just what do we mean by deaths occurring at random in time? Each interval, however short, must have a chance of containing a death equal to that of any other interval of equal duration. But the moment a death occurs, the number exposed is reduced by one, which immediately vitiates the randomness. The number of deaths, therefore, must be small compared with the number exposed, just as Poisson assumed, for the deaths to occur approximately "at random in time", which implicitly limits the law to small chances.

The Poisson series is the limiting form of the hypergeometric series when the chances are small.[3] Take an urn containing a few black balls distributed at random among many white, and scoop out enough at a time to include a few of the black. The probability of any number per scoop is always some term of the hypergeometric series. Now an exposure to risk is a sampling of this type, and so Poisson's Law should apply, approximately, to mortalities.

Let (D) be the number of deaths expected. Then the probability (P) that the actual number of deaths will be (d) is

$$P = \frac{D^d}{d!} e^{-D} \qquad (1)$$

where the constant $e = 2.718\ldots.$ is the Naperian base, and the symbol for the factorial expresses the continued product $(d! = 1.2.3.4\ldots.(d-1)d)$. This is Poisson's Law. It is expressed completely in terms of the expected number of deaths. Nothing else need be considered. The samples can differ in the numbers exposed, in their durations, or what not, provided their expected

3. This is proved in our paper "On the Dilution Method of Counting Bacteria", P. V. Wells and W. F. Wells, Jour. Washington Acad. Sciences, 11, 265-73, 1921.

deaths are the same, the distribution of actual deaths is given by the formula. Combine or subdivide our samples as we will, the expected number predicts the fluctuations of random sampling.

The Law is tabulated in Table 3, the expected deaths from 1 to 10 in the top row, the actual deaths from 0 to 18 in the first column at the left, and the corresponding calculated percentage probabilities in the body of the table. Imagine a sample exposure, say 300 risks for one year, could be repeated 100 times. If one death is expected per sample, the second column (which for brevity we shall designate by the head number 1, "column 1") shows that 37 of the samples will give zero actual

TABLE III.

Poisson's Law

Actual Deaths	EXPECTED DEATHS 1	2	3	4	5	6	7	8	9	10
	% of samples with expected (above) giving actual (at left)									
0	37	14	5	2	1					
1	37	27	15	7	3	1	1			
2	18	27	22	15	8	4	2	1		
3	6	18	22	20	14	9	5	3	1	1
4	2	9	17	20	18	13	9	6	3	2
5		4	10	16	18	16	13	9	6	4
6		1	5	10	15	16	15	12	9	6
7			2	6	10	14	15	14	12	9
8			1	3	7	10	13	14	13	11
9				1	4	7	10	12	13	13
10				1	2	4	7	10	12	13
11					1	2	5	7	10	11
12						1	3	5	7	9
13						1	1	3	5	7
14							1	2	3	5
15								1	2	3
16									1	2
17									1	1
18										1

deaths, 37 one each, 18 two, 6 three, 2 four, and all other numbers of actual deaths will occur in a negligible percentage of the samples. Another way of expressing the probability is to say that there is one chance in five (20%) of obtaining 3 actual deaths when 4 are expected (column 4, row 3).

Suppose we have an experience of 7 actual deaths, how many should we expect if the same exposure could be repeated? Comparing the probabilities of Table 3, in the row with 7 at the left ("row 7"), notice that the maximum, 15%, is found in column 7. Therefore, the most probable number of expected deaths is equal to 7, the number of actual deaths. The same proposition holds for every row of the table. Thus, Poisson's Law tells us to expect in the future the actual experience of the past if we have no *a priori* reason to expect something else. This is common sense. Nevertheless, it does not preclude the possibility of error in past experience, for this is but a random sample from a vast population of such risks. A glance at the table shows that it is almost as probable (14%) that 7 actual deaths will occur when the expected number is not 7 but is 6, or 8. Indeed, there is one chance in 50 (2%) of getting 7 deaths when only 3 deaths are expected. Such are the fluctuations of random sampling!

3. Errors of Sampling

In the ideal envisaged by the law, the same number of deaths is expected in each sample; but in practice the most we can expect is the same mortality ratio. Nor is this truly "expected". It is merely an index number used for comparison, and the bias due to its arbitrary base is all too evident at times. Now, obviously, it would completely upset the predictions in Table 3 to base them on any such wrong number of expected deaths. The proper number to take is the one truly expected, the most probable number, and the best estimate of this, *a posteriori,* is usually the actual number itself.

If the actual number of deaths taken is not the correct number to expect, this must in itself introduce an error into our predictions. For example, if the actual number is 5 and the true

number to expect is 8, Table 3 shows that by taking 5 as the expected number we would just double the predicted frequency (from 9% to 18%). But, unfortunately, we are never able to do better than estimate the number to expect. We must, therefore, predict the error of sampling on the basis of an expected number which is itself subject to an error of estimation.

It is customary to use the "probable" or median error to interpret the significance of an extra mortality. This "probable deviation" is defined in the Medical Impairment Study as two-thirds of the ratio of the mortality to the square root of the number of actual deaths, which is valid only when the actual is the correct number of expected deaths. Nowadays, most statisticians use the standard error, to avoid the constant 0.67449. For a Poisson distribution the standard error of sampling is simply the square root of the number of expected deaths. When this number is large, the distribution is practically normal (the simple "law of error"), and then the probable error is exactly the median error, than which one-half of the errors are less and one-half of them greater in magnitude. Only 31% are greater than the standard error (plus or minus). The border of significance is usually taken as twice the standard error, which is almost exactly three times the probable error.

To illustrate, the standard error of 9 expected deaths is its square root, or 3 deaths, and the probable error is $3 \times \frac{2}{3} = 2$ deaths. If the mortality expected with 9 deaths is 100%, the standard error is 33% and the probable error 22%. Now Table 3 shows that when 9 deaths are expected, the chance of obtaining more than 11 deaths is 7+5+3+2+1+1=19%, and 7 deaths or less is 12+9+6+3+1=31%, so that the chance of actual deaths falling without the borders 9 ± 2 is 19+31=50%. If we had included 11 deaths and excluded 7, the sum would have come out 48%. That is, about one-half of the samples fall inside the borders given by the probable error, and one-half of them fall outside, even for small numbers of expected deaths. For large numbers the proportions are almost exactly equal.

The probability of an extra mortality exceeding three times its probable error due to sampling is assumed to be about 2%,

or one chance in fifty, because this is the value given by Poisson's Law when we know the true number of deaths to expect, and when this number is large. The chance of a mortality being less than 100% by the same amount (or more) is assumed to be 2% also, but a glance at Table 3 shows that this exaggerates the chances for small numbers of expected deaths.

A more reasonable criterion of significance is suggested in Table 1. The number of actual deaths is tabulated beside the minimum significant mortality above 100%, and the maximum significant mortality below 100%. Before the experience the mortality expected was 100%, but afterwards the actual deaths are taken as the best estimate of the true number. Strictly, the table states that 100% is significantly different from the borders, which are assumed to be the correct mortalities to expect in the future. By expressing it this way, the table is made to read directly.

The borders of significance adopted in Table 1 are such that 95% of random samples are supposed to fall within them.[4] They merge into the borders given by $100(1 \pm 3 \times \text{probable error})$, when the number of deaths is large. To show their relation to each other and to Poisson's Law, the analysis of Table 4 is given for expected deaths 20 and less. The borders are expressed as actual deaths, instead of mortalities, to facilitate comparison with Table 3. Four borders are given for each number of expected deaths. The first (d_1) is the minimum above expected which is significant, and the second (d_2) is the maximum below expected, by Table 1. The third (d_3) and fourth (d_4) are the corresponding borders assuming three times the probable error as the criterion.

In the last four columns, the probabilities of actual deaths outside these borders have been obtained by adding up all the percentages in Table 3 from the actual numbers (d_1) or (d_3) down, and from (d_2) or (d_4) up. These are correct when the chosen numbers of expected deaths are correct but as any errors

4. The functions taken are $100/(1-t/\sqrt{d})$ and $100/(1+t/\sqrt{d})$, respectively, where d is the number of actual deaths, and t is taken (for $P = .05$ and $n = d-1$) from R. A. Fisher, Statistical Methods for Research Workers, 3rd ed., 1930, Table IV, p. 139, (Oliver & Boyd, London).

TABLE IV.

Comparison of Borders of Significance with Poisson's Law

Expected Deaths D	Significant Actual Deaths Min. d_1	Max. d_2	Expected plus minus Prob. Error x 3 Min. d_3	Max. d_4	Corresponding Poisson Probabilities of being More than d_1 %	d_2 %	Less than d_3 %	d_4 %
1	..	..	3	0	.0	8.0	.0	.0
2	20	0	5	0	.0	6.0	.0	.0
3	10	0	6	0	.1	5.4	.0	.0
4	10	0	8	0	.6	5.1	.0	.0
5	11	0	10	0	1.1	4.8	.0	.2
6	12	0	11	1	1.7	4.5	.0	.3
7	14	0	12	2	2.0	4.3	.0	.4
8	15	1	14	2	2.2	4.2	.1	.5
9	16	2	15	3	2.4	4.1	.2	.6
10	17	3	16	4	2.5	4.0	.3	.7
11	18	4	18	4	2.5	3.9	.3	.7
12	20	4	19	5	2.6	3.8	.4	8.
13	21	5	20	6	2.6	3.8	.5	.8
14	22	6	22	6	2.6	3.7	.5	.8
15	23	7	23	7	2.7	3.7	.6	.9
16	25	8	24	8	2.7	3.6	.6	.9
17	26	8	25	9	2.7	3.6	.7	.9
18	27	9	27	9	2.8	3.5	.7	.9
19	28	10	28	10	2.8	3.5	.8	1.0
20	29	11	29	11	2.8	3.4	.9	1.0

in our estimation of these will tend to bring the estimated closer to the actual numbers, they will lead us more often than not into that "fool's paradise" where we think we know more than we do. The more conservative borders (d_1) taken from Table 1 do not exaggerate the probable significance as much as do those (d_3) taken from the probable error. Both (d_2) and (d_4) appear to be too conservative, but there is little harm in such an interpretation of mortalities better than expected.

4. Mortalities Obey Poisson's Law

One cannot cease wondering about the remarkable simplicity of this law. By some trick of logic we seem to have derived

something from nothing. The numbers in Table 3 need not be deaths, they apply to all sorts of sampling problems. Frances Thorndike[5] has accumulated all the extensive distributions which have been published, such as death notices from the London Times, counting of alpha particles from radium, of bacteria, yeast cells, red blood corpuscles, lost articles, fires, incorrect reports, telephone calls for wrong number, wrong connections, and fifteen other telephone problems, army deaths from kick of a horse, etc. She concludes: "The applicability of the Poisson summation to a great variety of data is clearly indicated". Thus, experience shows a quantitative agreement whenever the conditions are such that it would be expected to apply. Bortkewitsch was so impressed by its purely numerical appearance that he dubbed it the "Law of Small Numbers"; but Poisson's Law is not limited to small numbers. Its essential characteristic is the smallness of the chance, such as death, which is being sampled. The law represents the ideal limit of uniformity in such samples, the "norm" from which departures are significant.

To show that the frequencies of death predicted by Poisson's formula do agree with the facts, we shall apply it to the detailed data of the Medico-Actuarial Mortality Investigation. In volume I, pages 96-97, are given the actual deaths by single policy years of the standard risks in 5 year age groups, and the expected deaths by the M A table based upon the same experience. We may, therefore, assume that the differences between the actual and expected deaths are due merely to the fluctuations of random sampling. Computing these differences for each pair of deaths in Table XII (except the synopsis), one-half of them should exceed their probable errors and one-half should be less. The actual numbers greater are shown by age groups in the second row of Table 5, and the percentages of classes greater are given in the bottom row. They tend obviously to the value 50%, as they should. Actually, 141 classes out of 276, or 51%, have deviations exceeding their probable errors. Even this slight discrepancy is in the right direction to be accounted for

5. "Applications of Poisson's Probability Summation", Bell System Technical Journal, 5, 604-24, 1926.

by the fact that the numbers of actual deaths must be slightly smaller than the numbers of policies, because of some multiple policies on single lives.

Including the synopsis tables, of all policy years, all ages at entry, etc., 169 classes out of 327, or 51.7% are greater than their probable errors. It is not necessary here to go into the systematic effects produced by the technique of graduation, which

TABLE V.

Classes with Deviations Exceeding their Probable Errors
(M.A.M.I. vol. I, pages 96-97)

Age (Years) Classes	15-9	20-4	25-9	30-4	35-9	40-4	45-9	50-3	54-6	57-9	60-2	63 & over
No. Greater than P.E.	11	11	14	13	11	13	11	10	8	18	11	10
Total No.	20	23	24	24	24	24	24	24	23	23	22	21
Ratio (%)	55	48	58	54	45	54	46	42	35	78	50	48

are quite evident in the summary classes. We have taken the detailed classes, to be relatively free from this bias. It may be remarked in passing, however, that the proper criterion for balancing "smoothness" and "fidelity", in E. T. Whittaker's probability method of graduation, is obviously that given by Poisson's Law. For, in view of the amount of our data, we should demand no more fidelity than can be obtained by random sampling, but have the right to demand that much.

To illustrate the effect of a systematic error on the frequencies in Table 5, apply the same analysis to the Medical Impairment Study basic mortality table, pages 162-164. Instead of one-half, over 90% of the actual differences are greater than their probable errors. This is due, evidently, to the fact that the numbers of deaths are very much less than the numbers of thousands of death losses, the unit amount being $1,000.

In volume II of the M.A.M.I., pages 66-72, the height and weight classes are given in great detail, which enables us to test Poisson's formula even better. Choosing weights within twenty pounds of the average, and heights from 63 to 66 inches, inclusive, (the group showing the best mortality) Table I gives

us 1,065 classes with expected deaths grouping 1 to 5. The actual distributions are shown in Table 6. They average 98% of expected mortality. The percentages in the last five columns approximate those in Table 1 quite closely. The actual differences between theory and practice are listed in the columns headed "Act. Dev." in Table 7. Their significant deviations are placed alongside at the left, their probable errors at the right, in the columns headed "Sig. Dev." and "P. E. ±", respectively. The former have been computed from Table 2, using the numbers of classes from Table 6 as "actual deaths", and multiplying the percentages by the observed percentages in Table 6.

Whenever the actual deviation in a class exceeds its probable error, an asterisk is placed beside it in Table 7. These vary in frequency from 33 to 58%, as shown at the bottom of the

TABLE VI.

Poisson's Law from M. A. M. I. Mortality Data

(1,065 Classes, vol. II, Table I, pages 66-72, incl.)

	EXPECTED DEATHS									
	1	2	3	4	5	1	2	3	4	5
Actual Deaths	No. with actual deaths at left					% of classes with actual deaths at left				
0	182	43	13	1	0	38.2	14.0	7.3	1.3	0
1	172	94	33	7	1	36.1	30.6	18.6	9.2	4
2	79	86	40	13	3	16.6	28.0	22.5	17.1	11
3	34	41	38	21	6	7.1	13.4	21.4	27.6	22
4	8	25	28	14	4	1.7	8.1	15.7	18.4	15
5	2	11	6	8	8	.4	3.6	3.4	10.5	30
6		6	11	6	2		2.0	6.2	7.9	7
7		1	5	4	1		.3	2.8	5.3	4
8			2	0	1			1.1		4
9			1	1	1			.6	1.3	4
10			1	0				.6		
11				0						
12				1					1.3	
Total	477	307	178	76	27	100	100	100	100	101

table. Out of the 50 classes, 22, or 44%, have deviations exceeding their probable errors. Adding the numbers in Table 6 in the classes asterisked in Table 7, we get 354, so that the weighted average is 33% of the 1,065 classes. Only two classes have deviations exceeding three times their probable error, or significant by Table 2, those with 2 expected, 3 actual, and 3 expected, 5 actual deaths, respectively. The latter is clearly a freak, for it is almost twice its significant deviation. There can be no doubt, however, that the actual deaths obey Poisson's Law.

Those familiar with modern statistical theory may be more impressed by the "Goodness of Fit" test of Karl Pearson, so the "divergences" are given in Table 8. In the last three rows are, respectively, the total divergences (χ^2), the numbers of independent groups (n), and the probabilities (P) that random samples obeying Poisson's Law will vary at least as much from expectation as do those in Table 6. The worst divergences are in the same two classes as in Table 7, which shows clearly the equivalence of the two methods. A value of P less than 5% is usually considered a significantly bad fit. This test, therefore, like the more elementary one of Table 7, demonstrates that these mortalities obey Poisson's Law.

5. Efficient Sampling

Another illustration of Poisson's Law is to determine how large a sample of exposed risks is required to reduce the sampling error to negligible proportions. A mortality ratio can never be relied upon closer than its probable error. But the number exposed is usually a hundred or more times the number of deaths per year. Now suppose that a truly random sample of the exposed be taken, for simplicity say one to every ten. Then there would still be over ten times as many sampled as the entire number of deaths, per year, at least up to age 50. This sampling would increase the error in the mortality ratio by such a small amount, compared with the probable error in the deaths, that for practical purposes it is quite negligible.

TABLE VII.

Significance of % Deviations from Poisson's Law

(From % Data of Table 6)

Actual Deaths	EXPECTED DEATHS 1			2			3			4			5		
	Sig. Dev.	Act. Dev.	P.E. ±	Sig. Dev.	Act. Dev.	P.E. ±	Sig. Dev.	Act. Dev.	P.E. ±	Sig. Dev.	Act. Dev.	P.E. ±	Sig. Dev.	Act. Dev.	P.E. ±
0	5.7	1.4	1.9	4.3	.5	1.4	4.4	2.3	1.4*		.5	.9		1	*
1	5.4	.7	1.8	6.3	3.5	2.1*	6.5	3.7	2.2*	8.6	1.9	2.3		1	3
2	3.7	1.8	1.3*	6.0	.9	2.0	7.2	.1	2.4	10.3	2.4	3.2	27	3	4
3	2.5	1.0	.8*	4.3	4.7	1.4**	7.1	1.0	2.3	12.7	8.1	4.1*	23	8	6*
4	1.4	.2	.4	3.3	.9	1.1	6.1	1.1	2.0	10.7	1.1	3.3	24	3	5
5	3.6	.1	.2	2.4	0	.7	3.6	6.7	.9**	8.7	5.1	2.5*	25	12	7*
6				2.1	.8	.6*	4.2	1.2	1.3	8.3	2.1	2.2	63	8	3*
7					0	.2	3.5	.6	.8	8.4	.7	1.8		6	3*
8							9.9	.3	.5		3.0	*		3	3
9								.3	.4		0	1.3		0	3
10								.5	.4*		.5	*		2	*
11											.2	*		1	*
12											1.2	.9*			

Classes with % Deviation Greater than Probable Error

	2	3	4	6	7
Total Classes	6	8	11	13	12
Ratio (%)	33	38	36	46	58
Mortality (%)	99	105	95	94	90

*Classes with Dev. (obs.%—%from Table 1) greater than Probable Error (Cols. headed ±).
**Classes with Significant Dev. (Act. Dev. greater than Cols. headed Sig. Dev.)

TABLE VIII.

Goodness of Fit of Mortality Data to Poisson's Law

(Data of Table 6)

Actual Deaths	EXPECTED DEATHS 1	2	3	4
	"Divergences"			
0	.24	.05	1.94	.16
1	.07	1.43	1.55	
2	.86	.10	.00	.31
3	.79	3.74	.09	2.55
4	.16	.26	.12	.05
5		.00	7.95	1.27
6			.46	.47
7		.83		
8			1.63	.68
9				
10				
11				
12				
Discrepancy (X^2)	2.13	6.41	13.74	5.49
n	4	6	7	6
P (%)	70	39	6	35

The saving of expense, however, is enormous. Most of the cost of a mortality investigation is spent on surviving risks, very little on the cards of the dead. Consider, for example, the basic data of the M A Table. Over half a million policies were included, but only twenty thousand, or 4%, were deaths. If all the claims are taken, but only every tenth policy among the others, the cost is reduced 86%. A part of this saving we can afford to use in obtaining added detail in the medical features of the study, and this information is many times more valuable

than the slight increase in precision obtained from the 432,000 cards.

Suppose N lives are exposed for t years, with d deaths. Place $M = d/Nt$, and let us sample $1/s = n/N$ of the risks. Assuming Poisson's Law for the sampling, the total % error in the mortality (M) is the square root of the sum of the squares of the % errors in d and in n, so that

$$\left.\begin{matrix}\text{Probable Error} \\ (\%\text{ of Mortality})\end{matrix}\right\} = \frac{67}{d^{\frac{1}{2}}}(1 + Mst)^{\frac{1}{2}} \qquad (2)$$

Thus, the effect of sampling 10% of the exposed (s=10), when the mortality is less than 1% per year, is to increase the probable error by a factor less than 1.05, less than 1.10 for 2 years, less than 1.22 for 5 years, and less than 1.41 for 10 years. The American Men Ultimate Table does not reach a mortality of

TABLE IX.

Increase in Probable Error Due to Sampling 10% of Exposed

(M. A. M. I., vol. I, p. 98)

Ages at Entry	First Policy Year Deaths	Sampling of Exposed 100% Probable (%)	10% Errors (%)	Actual Errors (%)
15–19	55	9.1	9.2	0.7
20–24	307	3.8	3.9	.9
25–29	396	3.4	3.5	.4
30–34	327	3.7	3.8	1.3
35–39	331	3.7	3.8	3.9
40–44	227	4.5	4.6	7.8
45–49	201	4.8	4.9	2.3
50–53	135	5.8	6.1	1.4
54–56	80	7.5	7.9	5.7
57–59	73	7.9	8.5	12
60–62	35	11	13	20
63–65	24	14	15	4
66–68	7	26	29	14
69–70	2	48	55	50

TABLE X.

Increase in Probable Error Due to Sampling 10% of Exposed

(M. A. M. I., vol. I, p. 99)

Ages at Entry	Fifth Policy Year Deaths	Sampling of Exposed 100% Probable Errors (%)	10% Errors (%)	Actual Errors (%)
15–19	25	14	15	26
20–24	162	5.3	5.9	1.0
25–29	241	4.3	4.8	1.6
30–34	239	4.4	4.9	2.0
35–39	207	4.7	5.4	4.5
40–44	195	4.8	5.7	2.2
45–49	184	5.0	6.2	16
50–53	108	6.5	8.8	4.0
54–56	69	8.1	12	8.0
57–59	64	8.4	13	7.7
60–62	37	11	19	4.1
63–65	20	15	29	14
66–68	5	30	63	42
69–70	1	67	152	100

1% until nearly age 50, the M A Table until age 49. For the first policy year it is under 1% up to age 55 for the A. M. and 53 for the M A. Since preliminary medical studies seldom cover an exposure of more than 5 years, it is evident that the increase in the probable error is negligible for standard risks at ages under fifty.

To show the effect of sampling 10% of the exposed upon the probable error of the mortality, equation (2) has been applied to the basic data of the M A Table, for the first policy year in Table 9, and for the fifth policy year in Table 10. The probable errors in % of mortality are given in the third columns, while in the fourth columns these have been increased by the factor $(1+10M)^{\frac{1}{2}}$ in Table 9, and by $(1+50M)^{\frac{1}{2}}$ in Table 10. In the last columns are given the magnitudes of the actual minus expected deaths, expressed as percentages of the actual deaths. It is evident that the increase in the probable error is quite

negligible at all ages for the first policy year, and at all ages under 50 for five years.

Even if we did not include all policies at ages 50 and over, but sampled only one in ten at all ages, the average increase in the probable error would not be serious. For the average mortality rate is 7.15 per thousand, and the average exposure is 5.62 years. Hence $(1+\text{Mst})^{\frac{1}{2}} = (1+.0715\times 5.62)^{\frac{1}{2}} = 1.18$, or an 18% increase in the probable error. This is equal to the increase in Table 10 at age 40. At age 45 in that table the increase is 25%, but even so, the actual error is over twice the probable error with the increase, so that such an increase is seldom important.

The can be little risk of serious error if a 10% sample is taken at ages under 50, while all at ages 50 and over, and all claims are included. This would save over 400,000 cards, which at 10 cents per card, would reduce the cost from over fifty thousand to less than ten thousand dollars. Of course, we have chosen a favorable example, for all the risks were standard. All substandard lives should be included until the nature of the risk is thoroughly understood. Indeed, considering the cost of ignorance to the company, there would seem to be little excuse for neglecting to study every claim from all points of view.

The usual method of reducing the cost is to cut down the issues exposed, for example, by sampling one year out of ten. It is much more efficient to sample the same number of cards from the entire exposure as described above. The relative efficiency of the proposed method is measured by the ratio of the probable errors of the mortalities. This ratio comes out roughly as $\sqrt{s}$. Thus, at the same cost, the proposed method is over three times as efficient as the usual method.

Sampling every tenth policy (say every number ending in zero) has the advantage of reducing somewhat the proportion of multiple policies on single lives, for they are often numbered consecutively. The method has been in routine use in our office only since the beginning of the year, so that it is premature to discuss its practice. We have hopes, however, that it will save

enough to make possible a fairly complete medical study of every claim.

6. The Experimental Method

This method consists of the attempt to isolate the causes of death by holding constant all possible factors except one at a time, and thus measuring the differential due to the remaining variable. Of course, this leads to small samples with large errors, but the hope of the method is that the exclusion of extraneous factors more than compensates for the increase in error of sampling. Just as the potency of an extract increases as it is purified, or the activity of radium increases, so the mortality due to a specific cause should increase differentially as we approach the isolated cause. If it does not, one of two interpretations must be made, either the cause itself is weak or there still remain contrary effects which are neutralizing that of the cause investigated.

As a practical illustration, let us compare the contribution of the Prudential to the Medical Impairment Study with that study, selecting the mortality of those with high blood pressure. The necessary data are collected in Tables 11 and 12. The Prudential mortalities are consistently less than those of the M.I.S., as shown by the observed differences in column (7), which are given by columns (4) minus (5). Columns (8) indicate that the only significant difference is the total in the bottom row.

Each border of significance in column (8) is the product of the value from Table 2, entered with the equivalent death of column (3), and the weighted average mortality, column (6). The equivalent death is the product of columns (1) and (2) divided by their sum. They are but slightly smaller than the Prudential deaths because these are less than 5% of the whole study. In such a case weighted mortalities are not really necessary, but they are given for a few classes to illustrate the example. Notice that double weight is given the Prudential experience, for it is included also in the joint experience. Such a procedure is appropriate for company practice. In fact, if selection differs widely from that of the group, a company's own

mortality can be taken as expected if its experience is not too small. Fortunately, there is usually not enough difference between the mortalities to make any practical change in the border of significance.

It must be kept constantly in mind that the correct expectation is not at all the one given by the index number "% of Expected". No one expects any longer 100% mortality in classes with high blood pressure, although Dr. Fisher had much difficulty in converting some recalcitrant souls twenty years ago

TABLE XI.

Comparison of Company Mortality with M. I. S.

(By Ages at Issue)

Ages at Issue	Actual Deaths Pru. (1)	M.I.S. (2)	Equiv. (3)	% of Expected Pru. (4)	M.I.S. (5)	W.Av. (6)	Differences (%) Obs. (7)	Sig. (8)	Ratio (9)
				344 X (X 1)					
15–29	5	20	4	141*	105*	112	36	178	.2
30–39	9	35	7	204*	129*	144	75	134	.6
40–49	2	140	2	36*	178	176	—142	1580	.1
50 & over	17	176	15	150*	150	150	0	82	.0
All	33	371	30	133*	153	151	—20	56	.4
				344 X (Y 1)					
15–29		10			85*		—85		
30–39		26			175		—175		
40–49	4	136	4	181*	245		—64	390	.2
50 & over	8	210	8	115*	162		—47	134	.4
All	12	382	12	107*	181		—74	116	.6
				344 X (X 2)					
15–29		24			116*				
30–39	3	90	3	226*	258		—32	640	.0
40–49	8	298	8	208*	261		—53	217	.2
50 & over	17	338	16	142*	189		—47	100	.5
All	28	750	27	157*	215		—58	86	.7
				344X (X 1) (Y 1) (X 2)					
15–29	5	54	5	96*	105*	100	—9	124	.1
30–39	12	151	11	178*	197	195	—19	131	.1
40–49	14	574	14	121*	231	226	—110	131	.8
50 & over	42	724	40	139*	170	168	—31	54	.6
All	73	1503	70	136	187	178	—51	43	1.2

*Mortality not significantly different from 100%.

TABLE XII.

Comparison of Company Mortality with M. I. S.

(By Policy Years)

Policy Years	Actual Deaths Pru. (1)	M.I.S. (2)	Equiv. (3)	% of Expected Pru. (4)	M.I.S. (5)	W.Av. (6)	Differences (%) Obs. (7)	Sig. (8)	Ratio (9)
				344 X (X 1)					
1–2	15	105	13	148*	137	137	11	75	.1
3–5	10	153	9	104*	162	158	—58	114	.5
6–10	8	104	7	182*	159	161	23	134	.2
11–19		9			169				
All	33	371	30	133*	153	151	—20	56	.4
				344 X (Y 1)					
1–2	7	106	7	113*	143		—30	133	.2
3–5	5	140	5	117*	196		—79	243	.3
6–10		124			244				
11–19		12			173*				
All	12	382	12	107*	181		—74	116	.6
				344 X (X 2)					
1–2	15	216	14	145*	185		—40	102	.4
3–5	9	263	9	136*	211		—75	162	.5
6–10	4	224	4	482*	257		225	409	.6
11–19		47			240				
All	28	750	27	157*	215		—58	86	.7
				344 X (X 1), (Y 1), (X 2)					
1–2	37	427	34	139*	160	158	—21	52	.4
3–5	24	556	23	117*	186	183	—69	77	.9
6–10	12	452	12	203*	222	222	—19	142	.1
11–19		68			213				
All	73	1503	70	136	187	178	—51	43	1.2

*Mortality not significantly different from 100%.

to this attitude. But to estimate significance we must assume approximately, at least, the correct expectation, and for this, in Tables 11 and 12, we have taken the weighted average mortality.

The cumulative effect of adding experience is shown by this example beautifully. To make it evident at a glance, the ratio of column (7) to (8) is given in the last column (9). This ratio is unity at the border. While not a single difference is significant in either table except the total, the predominance of

the negative sign in column (7) proves unmistakably that there is a systematic effect. It is unnecessary to consider this separately, however, for it is just this effect which makes the total significant.

The ratios are collected together in Table 13. To help visualize them, remember that the probable error gives a ratio about .3, so that if the differences were due to sampling one-half of the ratios would be above .3, instead of 16 out of the 28 in Table 13. Notice that not a single subclass is responsible for the total significance. The chief contributor is that for 3-5 policy years, which is most consistently negative for all three

TABLE XIII.

Summary of Ratios of Significance

(From Tables 12 and 13)

		Ages at Issue				Policy Years		
344 X	All	15–29	30–39	40–49	50 & over	1–2	3–5	6–10
X 1	.4	.2	.6	.1	.0	.1	.5	.2
Y 1	.6			.2	.4	.2	.3	
X 2	.7		.0	.2	.5	.4	.5	.6
Total	1.2	.1	.1	.8	.6	.4	.9	.6

systolic subclasses. Ages 40-49 are of next importance. It is quite evident, from Table 13, that the significance is cumulative in some age and policy year classes, but not at all in others. Thus, the total (.8) for ages 40-49 is much greater than any subclass, while at the younger ages the totals are less. In the synopsis column, for all ages and policy years, the significance increases with increasing hypertension, but there is also a slight degree of neutralization in the total.

In other words, the Prudential experience with class 344X is consistently better than the M.I.S., especially in policy years 3-5 and at ages 40-49. It is just at these ages where the M.I.S. mortality is at its peak, while for these policy years our selection seems unduly severe.

So much for the differences. When the mortalities themselves are considered, a direct comparison with Table 1 shows that the

asterisked values in columns (4) and (5) are not significantly different from 100%, the expected mortality for standard risks. Strictly, Table 1 assumes that the borders are the correct mortalities to expect, and that 100% is significantly different from them.[6] Again it is found that it is only the total mortality for class 344X, a class with 73 deaths, for which the Prudential experience is sufficient to assert that it is significantly substandard. Every subclass but one by policy year, however, is significantly substandard in the M.I.S., and all age groups except the youngest. Nevertheless, the latter suggests that the selection against these young risks for hypertension may be too severe, although it would be justifiable to wait for sufficient experience to render the mortality significant.

In Table 14 are compared the standard and substandard groups of 344Q. The two combined give only 62 deaths, but the analysis shows very definitely that the results are significant. The standard group shows (column 4) two significantly high subclasses, ages 30-39, and policy years 3-5, by Table 1. The substandard group (col. 5) shows two also, ages 50 and over, and policy years 1-2. Both groups are significantly high in total mortality. Moreover, column 9 of Table 14 shows that some of the differences between the standard and substandard mortalities are significant, namely, ages 50 and over, and policy years 1-2, and especially the difference between the totals.

Having found these significant mortalities in Table 14, in spite of the smallness of the samples, the next question is: Significant of what? Here the medical sleuth must begin to examine the subclasses in detail, beginning with the claims, to see if he can unravel the mystery with a rational interpretation. Often the clue, once obtained, will suggest auxiliary studies which will prove or disprove the hypothesis. For such studies wealth of detail is required, and that is why the utmost completeness in the records should be insisted upon. Usually the mystery is quite simple, once the solution is obtained. It is my

6. That is why it does not agree with Table 2. Table 1 checks when Table 2 is multiplied by the ratio of Table 1 to 100, and then added to 100.

TABLE XIV.

Comparison of Standard with Substandard

(Prudential Contribution to M. I. S.)

Ages at Issue	Actual Deaths Std. (1)	Sub. (2)	Equiv. (3)	% of Expected Std. (4)	Sub. (5)	W.Av. (6)	Differences (%) Obs. (7)	Sig. (8)	Ratio (9)
				344 Q					
15–29	2			107*					
30–39	13	3	2	344	909*	450	—565	4040	.1
40–49	9	2	2	117*	333*	156	—216	1400	.2
50 & over	20	13	8	115*	451	247	—236	205	1.2
All	44	18	13	143	446	237	—303	142	2.1
Policy Years				344 Q					
1–2	17	10	6	119*	461	246	—342	258	1.3
3–5	24	6	5	200	375*	235	—175	292	.6
6–10	3	2	1	78*	741*	343	—663	..	
11–19									
All	44	18	13	143	446	237	—303	142	2.1

*Mortality not significantly different from 100%.

firm belief that such realistic study of small samples in sufficient detail offers the best prospect of a successful solution of selection problems.

In concluding I wish to express my gratitude to Dr. Patton for his constant interest, advice, and encouragement, and to thank Dr. Brown, Dr. MacKenzie and Dr. Dewis for their helpful suggestions.

DR. WELLS—Mr. President, I want to express my appreciation for the honor of addressing this Association. I have been amused myself with the way in which I learned Poisson's Law of Small Chances. When I was at the Bureau of Standards in Washington, my brother was a bacteriologist in the hygienic laboratory, and every time he returned from the field he had some new problem in bacteriologic statistics, the mathematical side of which he expected me to solve. I very soon revealed my ignorance, but when he disappeared, I looked it up hoping to be ready for him next time, and then when he returned, it

didn't take that bacteriologist long to show the limitations of mathematics.

Eventually we learned a new theorem—so we thought, and we published it, only to find out later that is was Poisson's Law of Small Chances nearly one hundred years old. So you see that I am familiar with not being aware of its existence. It was some balm to our pride that we are not the only ones that did that trick. Several others have done exactly the same thing, and so I wasn't surprised when I came to an insurance company and found that they were not thoroughly aware of it either.

DR. CHRISTIERNIN—Dr. McCrudden of the New England Mutual will open the discussion.

DR. MCCRUDDEN—The invitation to discuss Dr. Wells' paper, one of the outstanding contributions to mortality investigation, is a distinct honor.

The first important contribution to mortality investigation was the discovery of a technique for investigating group mortality. In 1892, in a paper read before the Actuarial Society of America, McClintock said,

> "Concerning future investigations of mortality experience . . . . . the following suggestions may not be unworthy of consideration. Let each life, or each policy, be represented by a separate card . . . . . Let each card indicate the amount assured, a description of the policy, and as many more particulars as possible concerning the physical condition and family record of the individual . . . . . With cards thus prepared it is possible to make a separate examination of the expected and actual loss upon any collection of cards which it may be found convenient to put together."

The next important event was the use of McClintock's technique. In a small way McClintock, himself, had used it before 1890; and one or two medical directors used it shortly there-

after. But it did not come into general use for some time. In a paper read before the Actuarial Society in 1900 McClintock said,

> "The chief object of this paper is to suggest that actuaries . . . . . devote attention to . . . . . special investigations by which the actual results on a given fraction of business are compared with an ideal standard . . . . . It is more important . . . . . to learn how fisherman compare with farmers, how physicians compare with manufacturers . . . . . and the like than it is to gather together all these heterogeneous materials into one grand average in the form of a new life table . . . . . The object for which these special investigations are needed is to assist the companies in the selection of their risks by the increase of their knowledge of the various circumstances which indicate or affect longevity . . . . . To carry out such special investigations properly, it is necessary to segregate a certain class of risks and to determine whether it is on the whole a class of good risks."

The results of such an investigation, begun in 1901, were published—"The Specialized Mortality Investigation"—in 1903.

The next event of importance was the use of McClintock's technique in the more extensive "Medico-Actuarial Investigation" begun in 1908 and 1909 and reported on from 1912 to 1915.

Up to 1913 large numbers were considered a necessary requirement for dependable results. The material for the Medico-Actuarial Investigation, for example, representing the combined experience of 43 companies, consisted of about 2,000,000 policies. But no sooner was the first volume of the "Report of the Joint Committee" off the press than Dr. Dwight's demonstration of the dependability of results based on small numbers, the next important advance, took place.

As investigations of small groups increased in numbers and the companies became more and more impressed with their

value, the problem of the optimum size for a group naturally arose. His clinical training inclines the medical man to stress the medical accuracy associated with homogeneity and small numbers; his actuarial associations incline him to stress the mathematical accuracy associated with large numbers. In 1913 Rogers said,

> "When I first began to interest myself in the subject, I remember that among the first questions that came across my mind was, what should be the size of the group in order to make it dependable."

But Rogers could not get a satisfactory answer to this problem and neither could Dr. Dwight. Dr. Dwight's demonstration of the dependability of results based on small numbers was qualitative. The real problem is a quantitative one. How dependable are the results in any given case? Dr. Wells has shown us how to solve this problem.

Dr. Wells' figures ought to prove especially helpful in appraising the value of such functional tests for selection as the cardio-respiratory test, the blood-sugar tolerance test, and other similar tests. In investigating the results of such tests we may group our material with respect to the nature and extent of the impairment which made the special test necessary. Was the special circulatory test made because of advanced blood pressure, because of a heart murmur of one kind or another, because of rapid pulse, or because of arrhythmia; if so, how much did the blood pressure or pulse rate deviate from the normal? Or we may group with respect to the response to the test. Was the response to the test hyperactive, hypoactive, or atypical? Medical accuracy calls for as many and as homogeneous groups as is consistent with dependability. How small and homogeneous can we make a group or, better, how dependable is a group of so and so many? And Dr. Wells answers this question.

While I have no criticisms to make, it is in order to call attention to three groups of pitfalls in the too literal application of Poisson's Law.

The first, relating to group membership, and covering questions regarding the homogeneity, representativeness, and completeness of any group under investigation, to the influence of company selection, to anti-selection, to distortion due to misleading averages, and to improper standards, has been so thoroughly discussed by Mr. Marshall of the Provident Mutual at the 1932 meeting of the Actuarial Association that I cannot do better than to refer to that discussion.

The second has to do with the deaths. We might study the influence of hours of sunshine or hours of rainfall on the rate of ripening of apples in an orchard by counting the number of supposed windfalls every morning under a sample of ten trees. But if a boy with a stick should pick some of these ten trees to operate on forces of two different kinds would have been at work and the happenings—apple dropping in this case—would not have been truly random as required by Poisson's Law. Now, similarly, two kinds of forces influence life duration: First, certain determinate attributes such as age, sex, personal and family history, structural, functional, intellectual, and social personal characteristics, and environment on the one hand; and, second, certain indeterminate accidentals such as casualties from fire, murder, automobiles, etc. In a large group of deaths the total potency of the indeterminate accidentals is so small that the mortality is practically a function of the determinate attributes. But in a small group the potency of the accidentals may predominate. In such a case are the happenings as a whole truly random as required by Poisson's Law?

The third pitfall has to do with the importance attaching to the happening of a long chance in a specific case. It may be mathematically sound to say, for example, that there is but one chance in, say, a hundred or more that a patient will die during an operation for, say, tonsillectomy. But *there is one* chance and that particular chance may happen in the case of your own child. In other words, chances may have qualitative as well as quantitative implications; after computing quantitatively the value of a chance we must still appraise its qualitative significance in any specific case. In our investigation of the mortality of those

insured after a blood-sugar tolerance test the first two deaths were casualties though the chances against such a happening were nearly 200 to 1.

Nothing that I have said in the way of warning as to the application of the Law of Small Chances detracts from the fundamental importance of Dr. Wells' contribution.

DR. CHRISTIERNIN—Dr. Lotka will continue the discussion.

DR. LOTKA—Dr. Wells himself pointed out that the principle upon which the border is chosen, 75%, is arbitrary or conventional. Now, in view of that fact, we may very well ask, "Could we do better than conventional? Could we at least indicate a principle which is something better than the conventional standard?" I think such a principle can be indicated, but I am going to extend no hope of solving the problem. Remember what it is we are beginning to do. We want to balance our action so that losses incurred by putting up the barriers too high and losses incurred by letting them down too low may be avoided and a balance struck in the best possible way.

Now, I believe it is rather obvious that we can't do that by just guessing at 5%. It is very unlikely that the 5% exactly hits the mark. As I said a moment ago, the chances of solving that problem, finding just the right mark, seem to be very small indeed and therefore we do fall back upon this conventional standard, but the reason I raise this question is this: What are we going to do in borderline cases, in cases that fall near the borders set by Dr. Wells? Are we to disregard entirely indications of a substandard risk when it does not quite reach the border set forth from the tables? I believe the general feeling will be there that we want to be conservative as a rule, even though we don't quite reach the danger point indicated by the tables. I believe that as a rule we should err on the conservative side. In other words, you might put it this way, that the border established should be used by us as a servant, as a guide. It will make a very excellent servant. I don't think we want to make it our master.

I can only repeat what has been said before, that we are very much indebted to Dr. Wells for causing us to think along these lines, and it is to be hoped that the subject will be further developed and put into use.

Dr. Wells—The paper, after winding up, has a paragraph on what to do after you find a significant mortality. That's only one paragraph in the paper, but it is the longest paragraph in the paper. Really the sampling problem is the smallest part of it, as all Medical Directors know. You have only begun the job. When you have the suggestion that you have got a significant mortality then you have got to go back and study the sample and try further experiments, etc., and when you do that, you find out that the sampling side of it is decidedly the least. So I am very glad that Dr. McCrudden reminded us of that side of the story.

I was glad that Dr. Lotka emphasized the essential problem of the border. It is a very big problem. It is constantly facing the Medical Directors. However, as he says, we are far from having any consistent policy. I am afraid that after you have examined these significant mortalities, probably more often than not you will be led by the test for significance to a disappointment that the cause of the significance is rather disheartening, but that will have a valuable result in that it will point out to you where you are making your mistakes all the way through, and it is only by finding those mistakes by such suggestions that you can make your premise.

Dr. Christiernin—Owing to Dr. Dublin's absence from the city, his paper will be put over until tomorrow morning. Dr. Carber, however, has consented to read his paper in place of the one by Dr. Dublin, on "Coding Methods Employed by the Mutual Life Insurance Company of New York".

A NEW GENERAL CODE OF IMPAIRMENTS

By Frank H. Carber, M. D.

Associate Medical Director, Mutual Life Insurance Company of New York.

The efficiency of a code system for purposes of communication or statistics is in direct proportion to the extent of available detail. Prior to 1902, at which time the first official list of about 100 impairments was compiled in code form, the M. I. B. functioned as a simple signal of warning. Gradual growth to its present capacity of more than 300 codes has broadened its field of usefulness so that it is now an effective general source of information and basis of statistical record, and is essential to satisfactory underwriting.

The large volume of correspondence through the Bureau, which is still necessary to meet our demands for more specific information, seems to justify the construction of a code system which will develop a much greater degree of detail, and at the same time enable us to subdivide our statistical data into smaller groups which may be isolated as desired, or reassembled into large groups whose components are properly selected and controlled.

We have incorporated the 1929 International Causes of Death and the 1925 Code, practically intact, as the basic structure upon which our detail is elaborated so that data already classified in our files may continue to be readily available without confusion and the scope of the system may include Death and Disability Claims and all other forms of insurance data. With the exception of occupations, for which we already have a satisfactory classification, provision is made for all non-medical impairments, and in the medical field, vague and indefinite symptom complexes are isolated from clear cut clinical entities so that separate records may be kept of both, and our statistics may be protected against avoidable inaccuracies.

FIGURE I.

0 MISCELLANEOUS
01 Miscellaneous Impairments
02 Miscellaneous Injuries and Diseases
03 Benign Tumors
04 Cancer and other Malignant Tumors
05 Hernias
06 Poisons
07 Family History
08 Anatomic Localization
09 Miscellaneous Operations and Treatments—Special Diagnostic Methods

1 INFECTIOUS
10 Infectious Diseases
11 Syphilis (Understandard)
12 Syphilis (Understandard)
13 Syphilis (Understandard)
14 Syphilis (Understandard)
15 Syphilis (Understandard)
16 Gonorrhea
17 Tuberculosis
18 Parasitic Diseases
19 Special Infections

2 NERVOUS
20 Nervous Impairments
21 General and Infectious Nervous Diseases
22 Diseases of Brain and Meninges
23 Diseases of Spinal Cord
24 Diseases of Nerves
25 Nouroses, Psychoneuroses
26 Psychoses
27
28
29 Operations, Nervous System

3 CIRCULATORY
30 Circulatory Impairments
31 Heart Murmurs
32 Electrocardiographic Impairments
33 Teleradiographic Impairments
34 Diseases of Heart and Pericardium
35 Diseases of Arteries and Veins
36 Diseases of Blood
37 Diseases of Lymphatics and Lymph Nodes
38 Diseases of Spleen
39 Operations, Circulatory Organs

4 RESPIRATORY
40 Respiratory Impairments
41 Diseases of Nose and Throat
42 Diseases of Accessory Sinuses
43 Diseases of Larynx
44 Diseases of Trachea and Bronchi
45 Diseases of Lungs
46 Diseases of Pleura, Mediastinum and Diaphragm
47
48
49 Operations, Respiratory Organs

5 DIGESTIVE
50 Digestive Impairments
51 Diseases of Mouth and Salivary Glands
52 Diseases of Esophagus and Stomach
53 Diseases of Appendix and Cecum
54 Diseases of Intestine
55 Diseases of Rectum and Anus
56 Diseases of Liver and Gall Bladder
57 Diseases of Pancreas
58 Diseases of Peritoneum
59 Operations, Digestive Organs

6 GENITO-URINARY
60 Urinary Impairments
61 Genito-Urinary Impairments
62 Diseases of Kidney and Ureters
63 Diseases of Bladder
64 Diseases of Urethra
65 Diseases of Prostate and Seminal Vesicles
66 Diseases of Testes and Spermatic Cord
67 Diseases of Penis, Scrotum and Perineum
68
69 Operations, Genito-Urinary Organs

7 GYNECOLOGIC, OBSTETRICAL, BREAST
70 Gynecologic Impairments
71 Diseases of Uterus
72 Diseases of Ovaries, Tubes and Ligaments
73 Diseases of Vagina, Vulva and Perineum
74 Obstetrical Diseases
75 Diseases of Breast
76
77
78
79 Gynecologic, Obstetrical and Breast Operations

8 SPECIAL
80 Eye and Ear Impairments
81 Diseases of Orbit and Appendages of Eye
82 Diseases of Eye
83 Diseases of Ear
84 Diseases of Skin, Hair and Nails
85 Diseases of Deficiency
86 Diseases of Metabolism
87 Diseases of Endocrines
88 Diseases of Allergy
89 Operations, Eye, Ear and Skin

9 BONES, JOINTS, MUSCLES
90 Impairments
91 Deformities and Malformation
92 Diseases of Muscles and Tendons
93 Fractures
94 Diseases of Bones and Cartilages
95 Diseases of Joints and Ligaments
96 Diseases of Bursæ
97
98
99 Operations, Muscles, Tendons, Bursæ and Joints

FIGURE I—Continued.

CLASSIFIED ACCIDENTS

A–00 Miscellaneous
A–01 Accidents, Animal
A–02 Accidents, Asphyxiation
A–03 Accidents, Athletics
A–04 Accidents, Automobile
A–05 Accidents, Aviation
A–06 Accidents, Burns, Explosions
A–07 Accidents, Climate
A–08 Accidents, Drowning
A–09 Accidents, Electricity
A–10 Accidents, Falls
A–11 Accidents, Firearms
A–12 Accidents, Machine
A–13 Accidents, Mines, Quarries
A–14 Accidents, Poisons, Industrial
A–15 Accidents, Poisons, Non-Industrial
A–16 Accidents, Railroad, Elevator, Vehicle
A–17 Accidents, War
A–18 Suicide
A–19 Homicide

A–20—A–29 Available for Expansion
A–30—A–39 Available for Expansion
A–40—A–49 Available for Expansion
A–50—A–59 Available for Expansion
A–60—A–69 Available for Expansion
A–70—A–79 Available for Expansion

INSURANCE

A–80 Miscellaneous
A–8001 Disability Claim Allowed
A–8002 Confidential Information
A–8003 Occupation Unhealthful or Hazardous
A–81 Non-Conformity, six times a year
A–82 Non-Conformity, twelve times a year
A–83 Non-Conformity, weekly
A–84 Non-Conformity, two or three days, not oftener than three times a year
A–85 Non-Conformity, excess of above
A–86 Non-Conformity, steady free user
A–87 Non-Conformity, has taken cure
A–88 Non-Conformity, no details
A–89 Ineligibility
A–90 Insurance Hazard
A–91 Moral Hazard
A–92 Speculation
A–93 Environment
A–94 Industrial Grade
A–95 Informality
A–96 Aviation
A–97 Change of Residence
A–98 Disability Coverage Questionable
A–99 Reduced Amount of Insurance

B–00—B–09 Available for Expansion
B–10—B–19 Available for Expansion
B–20—B–29 Available for Expansion
B–30—B–39 Available for Expansion
B–40—B–49 Available for Expansion
B–50—B–59 Available for Expansion
B–60—B–69 Available for Expansion
B–70—B–79 Available for Expansion
B–80—B–89 Available for Expansion
B–90—B–99 Available for Expansion

(4)

The Key codes of two digits each have thirty subdivisions which are appended to the right as two additional digits to form the completed code of four digits. The non-medical codes are all preceded by the letter A.

FIGURE II.

7300 DISEASES OF VAGINA, VULVA AND PERINEUM (Con't)

7325 Benign Tumor or Cyst of Vulva
7326 Cancer or other Malignant Tumor of Vulva
7327 Hydrocele of Vulva
7328
7329

7400 OBSTETRICAL IMPAIRMENTS
7401 Pregnancy (664)
7402 Abortion, Miscarriage (661)
7403 Missed Abortion
7404 Threatened Abortion
7405 Incomplete Abortion
7406 Multiple Pregnancy
7407 Toxemia of Pregnancy
7408 Pernicious Vomiting of Pregnancy
7409 Hypertension of Pregnancy
7410 Eclampsia, Convulsions of Pregnancy (665)
7411 Dystocia, Difficult Labor (662)
7412 Prolonged Labor, Uterine Atony or Inertia
7413 Premature Labor
7414 Dry Labor
7415 Polyhydramnios, Hydrorrhea, Gravidarum
7416 Pelvic Deformity, Contracted, Flat, Funnel Pelvis
7417 Abnormal Presentation, Breech, Shoulder, etc.
7418 Still-Birth
7419 Placenta Previa
7420 Adherent Placenta
7421 Premature Placental Separation, Accidental Hemorrhage
7422 Sapremia, Retained Secundines
7423 Post-partum or Puerperal Hemorrhage
7424 Puerperal Septicemia, Sepsis, Fever
7425 Puerperal Phlebitis, Milk-leg, Phlegmasia, Alba Dolens
7426 Puerperal Pyelitis
7427 Puerperal Insanity
7428 Chorioepithelioma, Chorioma, Deciduoma, Syncytioma
7429 Hydatidiform Mole, Hydatid Mole

7500 DISEASES OF BREAST
7501 Acute Mastitis
7502 Chronic Mastitis
7503 Chronic Cystic Mastitis
7504 Benign Tumor or Cyst of Breast
7505 Cancer or other Malignant Tumor of Breast
7506 Paget's Disease of Nipple
7507 Hypertrophy of Breast
7508 Abscess of Breast
7509 Galactocele
7510 Fistula of Breast
7511 Caked Breast, Milk Retention, Duct Occlusion
7512 Fissure of Nipples
7513 Congenital Defects, Supernumerary Breast or Nipple
7514 Injury to Breast
7515 Thelitis
7516

FIGURE II—Continued.

A9600 AVIATION (856)
A9601 Passenger only, transportation planes, 10 or less flights annually
A9602 Passenger only, transportation planes, 11 to 20 flights annually
A9603 Passenger only, transportation planes, 21 to 50 flights annually
A9604 Passenger only, transportation planes, over 50 flights annually
A9605 Passenger only, taxi flying, 10 or less
A9606 Passenger only, taxi flying, 11 to 20
A9607 Passenger only, taxi flying, 21 to 50
A9608 Passenger only, taxi flying, over 50
A9609 Owner, pilot
A9610 Owner, not pilot
A9611 Pilot, transportation plane
A9612 Pilot, private plane
A9613 Pilot, taxi plane
A9614 Pilot, aircraft corporation
A9615 Pilot, army, navy, marine
A9616 Reserve pilot, army, navy, marine
A9617 Pilot, Auto-gyro plane
A9618 Pilot, glider
A9619 Student pilot
A9620 Previous pilot license now expired, has not flown in 0 year
A9621 Previous pilot license now expired, has not flown in 1 year
A9622 Previous pilot license now expired, has not flown in 2 or more years
A9623 Has received instruction, never licensed, has not flown in 0 year
A9624 Has received instruction, never licensed, has not flown in 1 year
A9625 Has received instruction, never licensed, has not flown in 2 or more years
A9626 Balloonist
A9627 Dirigible pilot
A9628 Reserve, army or navy observer
A9629
A9700 CHANGE OF RESIDENCE IF IMPORTANT, FOREIGN RESIDENCE (843)
A9701 Not tropical, own health
A9702 Not tropical, health of family
A9703 Not tropical, pleasure
A9704 Not tropical, business
A9705 Tropical, pleasure
A9706 Tropical, business
A9707 Foreign born, visiting country of birth
A9708 Foreign residence
A9709
A9710
A9711
A9712
A9713
A9714
A9715
A9716
A9717
A9718
A9719
A9720
A9721
A9722
A9723
A9724

(73)

The completed codes give the detail of the thirty subdivisions of each key code.

The 1925 code numbers which have been retained also appear.

FIGURE III.

0200 MISCELLANEOUS INJURIES (880) AND DISEASES (Con't)

0223
0224
0225
0226
0227
0228
0229

0300 BENIGN TUMORS (150) CYSTS
0301 Granuloma
0302 Hematoma
0303 Fibroma (156) Keloid, Desmoid, Fibroid, Xanthoma, Myofibroma, Myxofibroma
0304 Lipoma, Fibrolipoma
0305 Adenoma (155) Cystadenoma, Cystic Adenoma
0306 Polyp, Polypoid Adenoma, Fibroadenoma, Adenofibroma
0307 Papillary Adenoma, Papillary Cystadenoma, Adenopapillary Cystoma
0308 Papilloma (158)
0309 Myoma, Leimyoma, Rhabdomyoma, Adenomyoma, Fibromyoma
0310 Osteoma, Exostosis, Callus, Osteochondroma
0311 Chondroma, Enchondroma, Chordoma
0312 Neuroma, Ganglioneuroma
0313 Angioma, Hemangioma, Cavernous Angioma, Fibroangioma
0314 Lymphangioma, Hygroma, Cystic Hygroma, Fibrolymphangioma
0315 Myxoma
0316 Odontoma, Adamantinoma, Dentigerous Cyst, Radicular Cyst
0317 Cyst, Cystoma
0318 Retention Cysts, Mucus Cyst
0319 Dermoid Cyst
0320 Hydatid Cyst, Echinococcus Cyst
0321 Sebaceous Cyst
0322 Fetal Adenoma
0323
0324
0325 This code has been elaborated for optional use
0326 on occasions when it may be desirable to report
0327 the exact type of Benign Tumor or Cyst.
0328
0329

0400 CANCERS AND OTHER MALIGANT TUMORS (151)
0401 Epithelioma (153)
0402 Basal-cell Epithelioma, Rodent Ulcer
0403 Squamous-cell, Prickle-cell Epithelioma, Acanthoma
0404 Adenœpithelioma, Adenocanthoma
0405 Chondrœpithelioma
0406 Endothelioma, Psammoma, Perithelioma, Cylindroma
0407 Carcinoma
0408 Medullary Carcinoma
0409 Schirrus Carcinoma, Fibrocarcinoma
0410 Adenocarcinoma, Alveolar, Acinar, Tubular, Carcinoma, Cystadenocarcinoma
0411 Papillary Carcinoma, Papillary Cystic Adenocarcinoma
0412 Sarcoma
0413 Spindle-cell Sarcoma
0414 Round-cell Sarcoma

(6)

Optional codes are available by which the completed code, which contains the essentials of a given impairment, may be built up to any desired degree of detail through the addition of one or more optional codes, all being inclosed in parentheses.

7505N—Cancer of Breast within a year (see Figure II.).

(7505N, 0410N)—Adenocarcinoma of Breast within a year.

(7505N, 0410N, 7929N)—Adenocarcinoma of Breast with radical removal within a year.

(7505N, 0410N, 7929N, 0908N)—Adenocarcinoma of Breast with radical removal and X-ray treatment within a year.

FIGURE IV. THE INDEX.

Pylorus (Con't)
PYLORUS, PYLORIC (Con't)
10 Stenosis 5221
10 Stricture 5221
10 Tumor (5218)
10 Ulcer 5216
PYONEPHROSIS 6206
PYOPERICARDIUM 3423
10 PYOPHLEBITIS (3521)
5 PYORRHEA 5101
10 PYOSALPINX 7212
10 Gonorrheal 1609
10 Puerperal (7212, 7400)
10 Ruptured 7213
10 PYOTHORAX 4627
5 PYREXIA 0112
5 PYRIDIN POISONING 0617
10 PYROMANIA 2514
5 PYROSIS 5001
10 PYURIA 6023

Q

Q–R–S
10 Abnormal 3220
10 Notched 3220
Prolonged 3217
10 Splintered 3220
QUARTAN
5 Fever 1802
5 Malaria 1802
* QUARRY ACCIDENT, SEE (A1307-A1308)
5 QUINIDINE POISONING 0624
5 QUININE POISONING 0624
5 QUINSY 4126
QUOTIDIAN
5 Fever 1802
5 Malaria 1802
Q WAVE, DEEP 3221

R

5 RABBIT FEVER 1021
RABIES 2114
5 RACHITIC DEFORMITY CHEST (9106, 8503)
10 RACHITIS (8503)
RADICAL
Amputation, Breast (7929)
10 Mastoid Operation 8910
10 RADICULAR CYST 0316

Rectum
10 RADICULITIS 2411
RADIUM
10 Burn (A0609)
Effects of Exposure 0218
5 Treatment (0909)
* RADIUS, RADIAL (0810)
* Head (0809)
* Neck (0809)
* Styloid (0811)
* RAILROAD ACCIDENT, SEE A1600
10 Spine 2321
5 RALES IN CHEST 4001
5 RANULA (5123, 0318)
10 RAPID PULSE, SEE 3010-3011
10 RAREFACTION BONE (9411)
5 RASH (8400)
5 Drug (8412)
Syphilitic, Macular 11-1503
Syphilitic, Papular 11-1504
RAYNAUD'S DISEASE (2505)
10 RECLUS' DISEASE 7503
5 RECTOCELE 7309
RECTOVAGINAL
10 Abscess 5811
10 Fistula 7311
RECTOVESICAL
10 Abscess 5810
10 Fistula 6327
RECTUM, RECTAL
10 Abscess 5517
Cancer (5509)
10 Cyst 5508
5 Disease 5500
Excision (5920)
10 Fistula 5512
5 Foreign Body 5507
10 Hemorrhage 5015
5 Impaction 5506
Inflammation, see Proctitis
10 Injury (5505)
10 Malformation 5520
10 Polypus 5521
5 Prolapse 5511
10 Perforation 5503
10 Traumatic (5503, 5505)
Resection (5920)
10 Rupture 5503
10 Traumatic (5503, 5505)
5 Spasm 5515
Stricture 5504
Syphilis 11-1519
Tuberculosis 1715

(91)

All impairments are recorded for an unlimited period unless they are preceded by the numerals 5 or 10, in which event they are not recorded for more than 5 or 10 years from the date of the impairment. When limitation of space makes further elimination necessary, the codes without time limit are given precedence for retention in the records, followed in turn by the 10 and 5 year codes which are eliminated in reverse order to conform to the space which is available.

Codes preceded by an asterisk (*) may only be used jointly; codes inclosed in parentheses are usually used jointly but may be used singly.

The senior code of any group determines the time limit and precedence of the entire group.

FIGURE V. THE MORTALITY CARD.

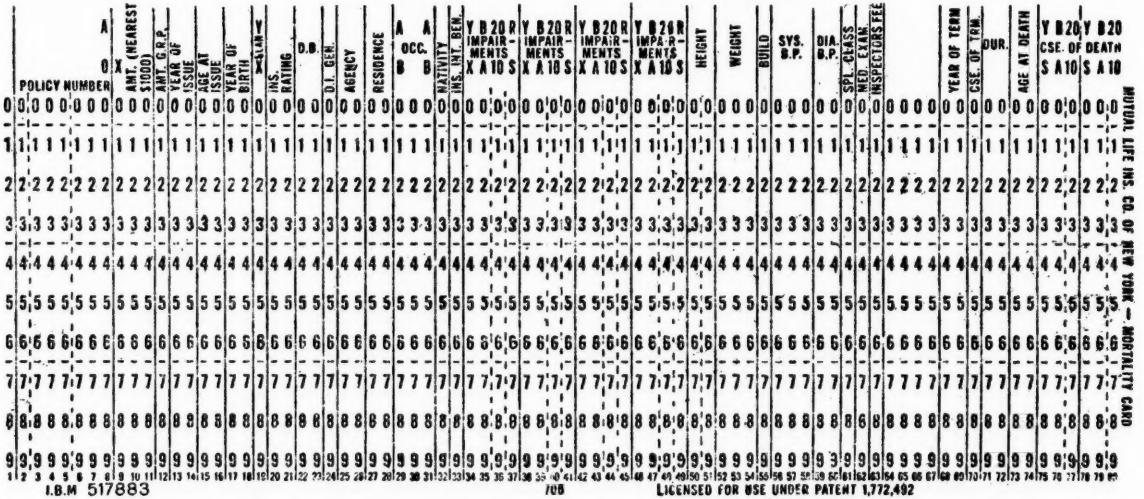

FIGURE V. THE MORTALITY CARD—Continued

Completed codes, including their time symbols, which are identical with those now in use, require a field of 4 columns which is our present space requirement for each code. The first two columns contain the two digits of the Key Code. The third column, by punching the 10 or 20 above, contains the 30 subdivisions which constitute the two right hand digits appended to the Key Code. The fourth column contains the time symbols.

41% of our records require 1 code.
30% of our records require 2 codes.
12% of our records require 3 codes.
17% of our records require 4 codes.

The system is therefore 41% effective in space allotted for 1 code; 71% for 2 codes; 83% for 3 codes.

Codes which are used jointly and inclosed in parentheses are so indicated on the card by punching the "Y" over column 1. The "X" over this column is the operator's "skip". "A" over column 2 is used with all non-medical impairments and "B" over this column provides for 100% expansion of the Medical Code. "R" and "S" over colunm 4 indicate respectively codes reported by another Company, and suspected.

This system has been in operation in our Bureau of Statistics for the past six months. Records are made of 64% of all applications, which is less than 1% increase above normal. There is an increase of 29% in the number of codes used and more than 90% normal efficiency of operation.

The Nomenclature which is applied wherever our clinical material presents sufficient specific detail, conforms to the new National Nomenclature, the Bellevue Nomenclature, and other classifications in general use by hospitals, clinics and other medical and health organizations with which interchange of data should be facilitated.

There is sufficient flexibility to permit contraction to limited space on the Hollerith Cards or expansion to express any desired degree of detail. There are ample provisions for future growth to keep pace with advances in our methods of selection.

For clerical efficiency of operation, an index of synonyms has been compiled upon the structure of the index of the 1929 International Causes of Death and a simple method has been devised to eliminate minor impairments of remote date for conformation to limited space on the Cards, and to retain for record the impairments of major importance.

DR. CARBER—I can't forebear remarking that this change in the order of our program is most timely. Here is a book full of codes of small groups to which to apply the Law of Small Chances.

I am sorry that we haven't enough of these code books available for general distribution. As you see, it is a loose-leaf book, eight by eleven, taking a standard size page. It has about 50 pages devoted to codes for small medical groups and about 20 pages for non-medical and an index of 100 and some odd pages. We have brought up several copies of this and Mr. Littlefield is in the back of the hall with them and will be glad to demonstrate them to any one who is further interested.

I have lantern slides of specimen pages which illustrate the various points that I want to bring out.

DR. CHRISTIERNIN—Mr. Wendell Strong, Associate Actuary of the Mutual Life Insurance Company of New York, will open the discussion.

MR. STRONG—Modern times have seen a great change in methods of doing nearly everything from earlier periods. This has been particularly in the line of specialization and the change is one that has been gaining momentum continually. In one sense it is an essential of what we speak of as "the machine age". From the times of our great-great-grandfathers, when, in this country at least, the man and the woman could do nearly everything necessary to a living, the man cultivating the fields, taking care of the animals, building his own house and digging his own well, and the woman not only making the clothes but spinning and weaving the cloth from which clothing was made, it has come to a time when the man perhaps can do only one thing, one motion in the making of a Ford car, but does that with a speed and certainty which were unknown and unthought of in the earlier time to which I have referred.

In all lines of endeavor and knowledge the tendency has been the same, from the general to the specific, then to the more specific, and again to the still more specific, and on and on.

This has been true with life insurance as well as in other lines. In the early days selection was exercised by having the applicant appear before the Board of Directors and they judged by his looks whether he was a good risk or not. Later there came medical examinations and the opinion of a physician on the examination. The work of a physician at the Home Office gradually developed into that of the medical director of today and the early crude method of selection into the highly specialized methods that we now have.

Dr. Carber's new code of impairments is a change along this line. There have been several changes of the code before; each, I believe, has resulted in being more specific than the former and the one we are discussing adds greatly in this line to the one that has been in use. Such a code has two different uses; it records impairments for the gaining of information and it also is the basis of future investigations of mortality under the various impairments.

The first of these, the gaining of information, has to do directly with present selection. I think it goes without saying that the more specific the information of the previous impairment is the more it aids one in selection. It can be told more readily whether the impairment is one that can be disregarded or needs to be investigated. The Medical Director who sees a code in a particular case knows better just what impairment he is dealing with. In selection this is an important advantage, and the more specific the coding can be made the better from this standpoint.

As regards investigation of mortality this also has to do with selection, but it is in the way of getting statistical information for future selection. Over thirty years ago Mr. Emory McClintock said that general mortality investigations were not the important things for the future. The important thing was the specialized investigation, which meant investigation by classes, and the more important classes from the standpoint of the life insurance companies are those having to do with impairments, that is, the classes to which the codes apply. This was true at the time of the Specialized Investigation which appeared in

1903, and which is now obsolete both for the reason that it refers to so many years ago and for the reason that most of the classes were not defined as we define them today.

It is fundamental that the classes shall not be so broad as to consist really of several sub-classes, which are essentially different. If, for instance, we investigated the mortality of the insured lives in a company which did business only in Canada and the tropical countries of South America and took the general mortality experience of that company, it would be worthless, since the Canadian mortality and the South American mortality could be expected to differ essentially and the final result would be very largely influenced by which section had the preponderance of the business.

In medical and other insurance impairments a similar thing is true. If we group under one impairment a number of essentially different classes our results are either worthless or worth much less than they should be. On the other hand, specialization can proceed too far, particularly so far as the statistical information for guidance in future selection is concerned. If we so subdivide classes that the numbers in each class are too small to get any adequate mortality experience on that class our mortality investigation will not do us much good. Of course, there is a remedy at hand if after the sub-division several closely related classes, where the impairment is very nearly the same, are grouped, so that the larger experience can be obtained, and perhaps in that way the damage of too great subdivision can be obviated. However, it is better when possible to avoid the necessity of this and have the classes sufficiently broad to give adequate numbers but have the results of the investigation of the mortality of such classes mean something. This would not mean, however, that in any case classes with fundamentally different impairments, even though they might occur under the same general name, should be grouped together.

While the combination into definite and homogeneous classes is desirable almost to the limit, it is not always possible to accomplish this result from the information furnished. Thus the information may not be more definite than that required

for the present M. I. B. code. This situation has been met by Dr. Carber in his code. He has the broad indefinite classes, which are not necessarily homogeneous, to cover the impairments which are not known with sufficient definiteness to assign them to definite or homogeneous classes. He also has the more definite classes for the cases where definite knowledge is at hand. Thus in the information for underwriters which comes through the code there may be in a particular case the broad class where there is not enough information obtainable to assign it to a definite class, or the case may have definite information which would automatically assign it to a definite class. As an illustration of what I mean by broad classes, not necessarily homogeneous in the sense of the impairments being of a similar kind in the matter of medical sub-division, and definite classes, I have selected the following:

In the 1925 code Nos. 337, 338, and 339 are "irregular pulse", under 5 per minute, 5 to 10 per minute, and over 10 per minute, respectively; however, where the real information as to the mechanism of the irregular pulse is at hand, showing in one instance a simple harmless sinus arrhythmia or in another instance a serious heart condition as is indicated by a total arrhythmia, these facts can be coded under different numbers and the underwriter has definite information for his guidance; also in future investigations the definite classes can be investigated.

In a transition like the present from the M.I.B. code to Dr. Carber's code it must, from the standpoint of the actuary, always be kept in mind that it will be some years before the experience will have accumulated and become sufficiently old for a mortality investigation to be made under the new code classifications. Under such circumstances it would be very unfortunate to adopt new classifications if they were to be such that it meant that the experience from the time of their adoption could not be taken out according to the old code because it would mean that for several years at least we could not add to our mortality experience that of the latest years of issue. Even

though these might not be enough to justify a separate experience, their value in being added to the old experience must not be overlooked. This has been met in Dr. Carber's code so that we need not give up the use of the new material concerning mortality under the old method of classification until we have accumulated sufficient new material to make an investigation by the new classifications worth while.

Fairly recently the 80 column card has been developed for the Hollerith machine and this is of great importance with reference to such a code for it is the 80 column card which has made the full use of the details in this code possible. It, however, can be used with the 45 column card if advantage is taken of its flexibility and the higher degrees of detail are omitted.

In the above I have spoken of the general method. Whether the classes used for the coding have been properly chosen is something regarding which the layman cannot well exercise judgment since medical knowledge is necessary to properly select and define these classes. Assuming, however, that this coding has been done properly, the change appeals to the actuary because it seems to him that it is in the right direction of greater sub-divisions and particularly of the definiteness which, where it is possible, brings things that are alike together into the same class and separates things that are unlike, although they may have the same general name, into different classes.

DR. CHRISTIERNIN—Dr. Weisse.

DR. WEISSE—Dr. Carber and Mr. Strong have covered the points in this paper so thoroughly that it leaves me very little to say.

During 1931 I followed with a great deal of interest the birth and gradual development of this excellent code of general impairments which Dr. Carber has developed.

At first sight the Code impresses one as possibly cumbersome, but when you become familiar with its workings you begin to

realize how easily the various details can be handled. It is divided and subdivided to a very large degree and it is this very refinement of detail which renders it so valuable. After a punched card has been prepared from an examination, under this Code, I cannot conceive the necessity arising of ever again removing the papers from the file for further information on that case.

With this Code in general use and with the cards punched there should be no difficulty in obtaining practical homogeneous groups whether for medical, actuarial, or other study. The classification of impairments is sufficiently detailed to allow for the study of small as well as of large groups which is something we have not been able to achieve before without hand sorting large numbers of cards or examinations.

And here let me emphasize the importance of the small group —reiterating what the late Dr. Dwight of the New England Mutual first said many years ago and what a number of others have said since.

A company using this Code on its own business can keep track of small groups and thereby with very little trouble after the punching of the cards study the trend of their business.

We have been punching cards under this Code now for over six months and find that it takes very little longer to prepare the cards than when using the M.I.B. Code as heretofore and we feel that it is entirely satisfactory.

SECOND DAY

DR. CHRISTIERNIN—We will now resume the program by calling upon Dr. Dublin to present his paper, which was postponed from yesterday, on the subject of "Medical Selection in Cases with a History of Pleurisy".

MEDICAL SELECTION OF CASES WITH A HISTORY OF PLEURISY

By Louis I. Dublin, Ph. D., Third Vice President and Statistician and Herbert H. Marks,

Metropolitan Life Insurance Company, New York City.

Pleurisy is rarely a primary disease. The best medical authority supports the view that it is usually secondary to such conditions as tuberculosis, pneumonia, influenza, rheumatism and typhoid fever. Yet in most insurance and even in clinical histories, there is no clue to the infection which precedes the pleurisy. And so the classification or fiction of primary or idiopathic pleurisy has remained. It is with the large group of persons reporting such histories that we are concerned in this paper.

The chief subsequent hazard presented by applicants who give such a history of pleurisy is pulmonary tuberculosis. At the 1924 meeting of this Association, Dr. Martin reviewed the important sources of a statistical nature which bore out this viewpoint. Similar investigations which have been carried out since Dr. Martin's report confirm this opinion although they are somewhat more optimistic regarding the prognosis of patients with a history of pleurisy. But in general the medical and insurance attitude toward the disease remains pretty much the same.

Yet, in spite of this doleful outlook we have insured such applicants, some even on standard plans and the results have not been disastrous. It should prove interesting to see the reasons for this. These applicants for insurance are, in some respects, self-selected. They come to us for examination after recovery, often after a lapse of many years since their illness. Barring exceptional cases, they are in good health, active and vigorous. It is from such a broad group that we are called upon to select those who are eligible for insurance.

In this paper we offer the experience of the Metropolitan Life Insurance Company on 6,264 white risks who were insured by the Company in the period 1912 to 1928 and who were traced to the policy anniversary in 1930. This group includes only cases of primary pleurisy so far as the record goes. We have excluded as carefully as we could all those cases where the pleurisy was stated to be merely a complication of some other condition. We have likewise excluded all those cases which were substandard for any other reason than pleurisy, whether because of occupation, overweight or other medical impairments. We have also kept out those cases where the examining physician reported some physical evidence of the pleurisy still present, such as adhesions, pleural thickening, poor lung expansion, friction rub, dullness and the like, and also those risks who reported either a family history of tuberculosis or coresidence with a person suffering from the disease.

We have investigated three types separately, namely, pleurisy with effusion, dry pleurisy and pleurisy without details as to kind. We must frankly recognize that these distinctions are not water-tight. In the first place, while we are dealing with so-called idiopathic pleurisy, there are undoubtedly many cases where the pleurisy stood out in the applicant's memory and not the pneumonia or other condition of which it was a complication. Secondly, in many pleurisies reported as dry there was undoubtedly some effusion which the attending physician did not tap but permitted to be absorbed and the applicant did not know of this fact. Thirdly, and what is most important is that in many cases of pleurisy without details and in some of the dry pleurisies, we are not concerned with pleurisy at all but merely with a chest pain, a pleurodynia, a myositis, an intercostal neuralgia, or a similar condition. In recent years, particularly, we have tried to get as full information as possible, but in only a relatively small number of cases are we successful. Our results, therefore, must be judged in the light of these unavoidable defects.

In our analysis we have considered the important factors of selection of these cases, namely, the time elapsed since the illness, the duration of the attack, the build of the applicant and the age.

We have studied the experience on males and females separately. We have combined standard and substandard cases on the assumption that the separate factors entering into the rating of these applicants would tend to separate out the substandard issues from the rest.

Our results are presented in the form of ratios of actual to expected deaths by the American Men Mortality Table. We have made comparisons both on a select and ultimate basis and on an ultimate basis, but we shall quote the ratios by the first of these.

Let us review, briefly, the results we have obtained:

1. Male Risks with a History of Pleurisy

a. Pleurisy with effusion.

Our group of 6,264 cases includes 424 male applicants who reported pleurisy with effusion. The exposed to risk amounts to 2,821 years of life and there have been 19 deaths. Measured by the American Men Table, the mortality of these cases with effusion has been 124.6 per cent. of the expected. Analysis of the experience according to the time elapsed since the issue of insurance shows that the highest mortality occurred in the first five years after issue. The mortality in this period was 142.1 per cent. of the expected, compared with 106.5 per cent. in the later insurance years. Considered by age, it was found that the experience on the younger lives, namely, at attained ages under 35, was more favorable than at ages over 35, but the number of deaths at these young ages was small and we are not inclined to place any reliance on this result. At the older ages the mortality was 151.5 per cent. of the expected, but these extremely poor results on older lives were concentrated in the first five insurance years in which the mortality was 219.3 per cent. of the expected.

Although the group is small, we found a definite relation of the length of the period intervening between the illness and application for insurance to the subsequent mortality. Those insured within two years of their attack had a mortality four and a

half times the expected, and those insured from two to four years after illness, nearly twice the expected. The illnesses occurring more than five years before application yielded results which were appreciably better than expected by the standard table used.

TABLE I.

Mortality of Risks with a History of Pleurisy with Effusion

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Time of Attack before Issue and by Broad Attained Age Groups.

Attained age Time before issue	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	2821.17	19	124.6	113.2	142.1	106.5
Under age 35	1535.66	5	83.2	74.5	73.3	104.2
Ages 35 and over	1285.51	14	151.5	139.0	219.2	107.3
Less than 2 years	302.00	7	469.8	434.8		
2–4 years	633.60	5	171.2	153.4		
5–9 years	885.24	2	42.0	38.2		
10 years and over	987.36	5	83.9	77.0		

Although the number of deaths is small, it is interesting to note that of the 19, five were due to tuberculosis and two to influenza or pneumonia.

b. Dry pleurisy.

The experience on men reporting a history of dry pleurisy has not been good. By the American Men Table the mortality has been 134.1 per cent. of the expected. This result is based on a total of 841 male lives, yielding an exposed to risk of 5,123 years, among whom there occurred 39 deaths. The mortality in the early insurance years in these dry pleurisies accounted for a large part of the excess mortality. The deaths in the first five years after issue exceeded the expected by 54.4

per cent., compared with only 10.7 per cent. in the sixth and later years.

The mortality on younger risks has been the least satisfactory, exceeding that expected by the American Men Table by nearly 60 per cent. at attained ages under 35, and this excess of mortality has prevailed both in the first five years following issue and in the later years. At older ages the mortality has been better, but still 20 per cent. in excess of the table. In this case, however, the excessive mortality occurred in the early insurance years in which the actual deaths were 146 per cent. of the expected. Those applicants whose pleurisy occurred many years before the insurance was issued had a better mortality than those with a history of pleurisy in the more recent past. While the volume of material was insufficient to give a clear-cut result, we find that the group granted insurance within two years of illness had a mortality 136.5 per cent. of the expected; those insured from two to four years, 262.7 per cent.; those five to nine years, 76.7 per cent.; and those over ten years, 101.7 per cent.

Analysis of our experience according to duration of illness indicates that this factor is not very important in dry pleurisies. Among risks whose illness occurred within five years of issue, those with illnesses lasting only one or two weeks had a somewhat lower mortality than those with illnesses lasting three weeks or more. The ratios of actual to expected deaths were 193.7 per cent. and 226.5 per cent., respectively. Among those risks whose illness occurred five or more years before issue, this relation did not hold and although the number of deaths was too small to put much reliance upon them, the fact that we got excessive mortality for cases reporting a short illness within five years of issue, leads us to lay more stress upon the recency of the attack in dry pleurisy than on the duration of the attack.

Of the 39 deaths which have occurred in this group, nine were due to tuberculosis. This number is approximately double that expected on the basis of the Company's experience on standard lives. All of these deaths occurred in the first five years after issue. Six of them were of applicants granted standard insurance and three substandard. There were also six deaths from

TABLE II.

Mortality of Risks with a History of Dry Pleurisy

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Time of Attack before Issue, Duration of Attack, and by Broad Attained Age Groups.

Attained age Time before issue Duration of attack	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	5122.58	39	134.1	121.3	154.4	110.7
Under age 35	2607.59	16	158.7	140.8	163.5	146.0
Ages 35 and over	2514.99	23	121.0	110.7	146.3	101.8
Less than two years	1043.16	7	136.5	122.6		
2–4 years	1227.61	16	262.7	235.6		
Less than five years						
Ill 1–2 weeks	1150.27	11	193.7	173.8		
Ill 3 weeks and more	645.41	7	226.5	202.3		
5–9 years	1439.56	6	76.7	69.4		
Ill 1–2 weeks	680.16	3	90.1	81.5		
Ill 3 weeks and more	569.24	2	63.9	56.5		
10 or more years	1392.57	10	101.7	92.7		
Ill 1–2 weeks	689.24	6	120.0	109.5		
Ill 3 weeks and more	512.66	3	90.4	81.5		

pneumonia or influenza, but there was no concentration of these deaths in the early years following issue.

c. Pleurisy without details as to type.

By far the largest number of these men with a history of pleurisy gave no details as to the type. This group numbered 4,095, or over 75 per cent. of the total, with an exposed to risk of 29,602 years. There were 191 deaths in the period under observation.

The mortality of this group was almost exactly that expected by the American Men Table, namely, 100.8 per cent. It was somewhat higher in the years immediately following issue of insurance, exceeding the expected by 10.7 per cent. In the sixth

and later years the mortality was only 91.4 per cent. of the expected.

The experience at younger ages—under 35 attained—was somewhat worse than the average for the group. In this broad age group the actual mortality was 109.1 per cent. of the expected by the American Men Table. Among these younger persons the actual mortality was in excess of the expected over the whole period; in the first five insurance years, by 5.5 per cent., and in the sixth and later years, by 16.6 per cent. Over age 35, the experience has been, on the whole, better. At these ages the deaths were 97.6 per cent. of the expected. There was an excess mortality among these older risks in the years immediately following issue—104.3 per cent. of the American Men Table. In the sixth and later years it was only 86.1 per cent. of that table.

Among these men with pleurisy histories of unspecified type, those whose illness was recent suffered a higher mortality than those whose illness occurred in the more remote past. Thus, for those insured within two years of illness, the actual deaths were 31.3 per cent. in excess of the expected. In this group the results were extremely bad in the first five years after issue, when the mortality was 176.6 per cent. of the expected. The mortality of these men was high regardless of age, but more so among the younger risks.

When the insurance was granted more than two years after illness, there was little difference in the mortality in several groups classified according to the time elapsed. Four such groups were distinguished, namely, two to four years, five to nine years, ten to fourteen years, and fifteen years or over. The mortality ratios in these groups, compared with the American Men Table, were 90.4 per cent., 95.1 per cent., 100.0 per cent. and 97.9 per cent., respectively.

The most significant feature of the experience on these risks was the effect of duration of illness on the subsequent mortality. We have distinguished three classes of durations—illnesses lasting three weeks or more, those lasting one or two weeks, and those of less than one week. We have investigated

this factor according to the time intervening between illness and application for insurance so that due allowance would be given to that element.

We found that in every group classified according to time since illness, those reporting long illnesses have suffered a higher mortality than those with very short illnesses and the consistency of these results offsets the fact that the number of deaths in some

TABLE III.

Mortality of Risks with a History of Pleurisy, Kind Not Stated

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Time of Attack before Issue, Duration of Attack, and by Broad Attained Age Groups.

Attained age Time before issue Duration of attack	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	29602.14	191	100.8	92.4	110.9	91.4
Under age 35	13284.01	57	109.1	98.3	105.5	116.6
Ages 35 and over	16318.13	134	97.6	90.1	114.3	86.1
Less than 2 years	3940.34	28	138.3	126.3	176.6	99.5
Ill less than one week	1309.34	5	75.2	68.6		
Ill 1–2 weeks	1729.08	12	135.6	123.5		
Ill 3 weeks or more	698.50	5	134.4	123.8		
2–4 years	7026.11	35	90.4	83.0	98.8	82.1
Ill less than one week	1602.46	6	69.0	63.1		
Ill 1–2 weeks	3302.30	14	76.1	69.9		
Ill 3 weeks or more	1702.59	11	120.9	110.6		
5–9 years	7539.48	41	95.1	86.7	81.7	109.0
Ill less than one week	1394.55	7	81.8	75.0		
Ill 1–2 weeks	3250.70	17	92.5	84.3		
Ill 3 weeks or more	2428.38	13	100.9	91.2		
10–14 years	4564.97	29	100.0	91.5	101.5	98.6
15 years or more	6204.78	55	97.9	89.6	125.9	76.0
10 years or more						
Ill less than one week	1417.39	12	102.9	93.2		
Ill 1–2 weeks	5119.13	36	88.2	80.5		
Ill 3 weeks or more	3288.52	30	119.0	108.7		

of the groups was small. Thus, in the case of illnesses occurring less than two years before issue, the mortality among those disabled three weeks or more was 134.4 per cent. of the expected by the American Men Table, but only 75.2 per cent. among those disabled for less than one week. Similarly, when the illness occurred two to four years before issue, the mortality among those ill three weeks or more was 120.9 per cent., compared with 69 per cent. among those ill less than one week; where the illness occurred from five to nine years before issue, the corresponding percentages were 100.9 per cent. and 81.8 per cent.; and where the illness occurred ten years or more before issue, 119.0 per cent. and 102.9 per cent., respectively. The above comparisons relate to short and long illnesses. Illnesses intermediate in length, namely, one or two weeks, generally produced ratios intermediate between those quoted.

If we disregard differences in the time elapsed since illness, we find that cases with illnesses lasting three weeks or more

TABLE IV.

Mortality of Risks with a History of Pleurisy, Kind Not Stated

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Duration of Attack and by Broad Attained Age Groups.

Duration of attack Attained age	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Ill less than one week						
All ages	5723.74	30	84.4	76.9	82.1	86.4
Under age 35	2607.76	10	97.7	87.8	74.6	141.2
Ages 35 and over	3115.98	20	79.0	72.4	87.0	73.5
Ill 1–2 weeks						
All ages	13401.21	79	91.4	83.5	112.0	72.7
Under age 35	5978.47	23	98.0	88.0	107.0	79.3
Ages 35 and over	7422.74	56	88.9	81.8	115.2	71.4
Ill three weeks or more						
All ages	8117.99	59	115.9	105.6	135.6	96.7
Under age 35	3694.69	17	117.8	105.4	142.9	64.8
Ages 35 and over	4423.30	42	115.1	105.7	131.0	103.7

suffered a mortality 115.9 per cent. of the expected by the American Men Table; those with illnesses lasting one or two weeks, 91.4 per cent.; and those with short illnesses of less than a week, 84.4 per cent. Both at ages under 35 (attained) and over that age, cases with long illnesses experienced the highest mortality. But it is particularly marked in the early insurance years and among young persons. Thus, the mortality at ages under 35 in the first five years following issue was 142.9 per cent. of the expected by the American Men Table. The mortality in the corresponding group who were ill only one to two weeks was 107.0 per cent., and those ill less than one week, 74.6 per cent.

We have also analyzed the experience on these cases with a history of pleurisy to determine the influence of weight on the mortality. Our findings were negative when no account was taken of age and length of illness. Further analysis, however, showed that among young persons there was a distinct relation between weight and the subsequent mortality among persons who suffered from pleurisy. This is particularly clear in cases with a long illness. Thus, the mortality of those men under age 35, who were ten pounds or more underweight, was 127.1 per cent. of the expected by the American Men Table, compared with 89.9 per cent. among those of normal weight (within ten pounds of the average). Although the number of deaths was small, the results were confirmed in the group ill only one or two weeks, in which the mortality was 108.8 per cent. for the underweights and only 92.0 per cent. for normal weights. Among older persons the relation of weight to mortality among risks with a history of pleurisy did not hold. Overweights did not show consistently better mortality results than underweights except at the younger ages. A moment's reflection will show that this is not unexpected because, as is well known, among young persons, overweights have a lower mortality than underweights, but among older persons this tendency is reversed.

Out of 191 deaths which have occurred in this experience, 36, or 18.8 per cent., were from tuberculosis. This is about 50 per cent. higher than that prevailing in the Company's Ordinary

Department. At ages under 35, 19 out of 58 deaths, or 32.8 per cent. of the total were from this cause. At ages 35 and over, only 17 out of 133 were from tuberculosis, or 12.8 per cent. of the total. Of these 36 deaths from tuberculosis, the

TABLE V.

Mortality of Risks with a History of Pleurisy, Kind Not Stated

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Weight Groups, by Duration of Attack and by Attained Age Groups.

Duration of illness Attained age Weight groups*	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Ill 1–2 weeks						
All ages						
Underweight	3852.22	17	76.8	71.0	102.7	52.5
Average weight	5703.06	35	98.6	90.4	141.4	59.4
Overweight	3837.08	27	93.5	85.2	82.0	103.5
Under age 35						
Underweight	2061.24	9	108.8	99.9	110.7	105.3
Average weight	2763.81	10	92.0	83.0	108.8	56.8
Overweight	1145.41	4	89.7	80.0	93.2	80.6
Ages 35 and over						
Underweight	1790.98	8	57.7	53.5	94.5	35.0
Average weight	2939.25	25	101.5	93.7	166.3	60.0
Overweight	2691.67	23	94.2	86.2	78.4	105.5
Ill three weeks or more**						
All ages						
Underweight	2195.38	15	125.0	114.7	134.5	115.7
Average weight	3203.30	24	109.0	99.1	114.2	104.3
Overweight	2013.71	15	113.7	102.6	132.4	93.9
Under age 35						
Underweight	1201.72	6	127.1	114.3	132.5	117.6
Average weight	1421.71	5	89.9	80.5	103.4	59.2
Overweight	644.40	2	79.4	69.9	104.2	—
Ages 35 and over						
Underweight	993.66	9	123.6	114.9	136.5	114.9
Average weight	1781.59	19	115.5	105.5	120.5	112.1
Overweight	1369.31	13	121.8	110.5	143.4	103.6

*Underweight—10 pounds or more underweight
Average weight—9 pounds or less underweight or overweight
Overweight—10 pounds or more overweight

**Excludes a few cases less than two years before issue

majority occurred within the first five years after issue of insurance; indeed, eight of them occurred within two years of issue. Half of them occurred among those insured within five years of their illness. Deaths from this cause were relatively more frequent among those with long illnesses. Most striking, however, was the incidence of tuberculosis in underweights; 16 of the deaths occurring among men ten pounds or more underweight at issue and 12 more among those less than ten pounds underweight. There was a decided concentration of these underweight deaths among younger persons.

Influenza and pneumonia together accounted for slightly more deaths than tuberculosis. There were 39 deaths from these respiratory conditions accounting for 20.4 per cent. of all deaths. Deaths from these causes were relatively more frequent than tuberculosis among older men than among the younger ones. All types of respiratory conditions, including tuberculosis, accounted for 60 per cent. of the mortality among younger persons, but only a little over 30 per cent. among older persons. Accidents accounted for most of the remaining deaths among young persons. Among older persons, as would be expected, a very large part of the mortality was due to chronic disease; there were 23 deaths from heart disease, nine from cancer, eight from cerebral hemorrhage, seven from angina pectoris and six from chronic nephritis. Together these diseases accounted for 40 per cent. of the deaths at ages 35 and over. This is about normal.

d. Risks with a family history of tuberculosis.

The experience on risks with pleurisy, of which we have been speaking, excludes those who reported tuberculosis in the family history or contact with the disease during the year preceding application. We were able to segregate the records of 278 such men with a history of pleurisy who also reported these tuberculous contacts, but who were otherwise standard in all respects. About 70 per cent. of them were granted insurance at standard rates.

The experience on these 278 men has been comparatively good. In the experience amounting to 2,196 years of life, there have

occurred only 13 deaths, or 76.7 per cent. of the expected by the American Men Table. This experience represents very careful selection. Evidence of this is found in the relatively large proportion of the experience on older lives. The small experience on younger men has thus far been good; among them only two deaths occurred, or 68.3 per cent. of the expected by the American Men Table.

TABLE VI.

Mortality of Risks with a History of Pleurisy, Kind Not Stated, Who Had a Family History of Tuberculosis

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Broad Attained Age Groups.

Attained age	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
All ages	2196	13	76.7	70.4	41.7	102.7
Under age 35	746	2	68.3	61.0		
Ages 35 and over	1450	11	78.5	72.5		

Of the 13 deaths recorded, four were from tuberculosis. There were also two deaths from pneumonia.

2. White Female Risks with a History of Pleurisy

We were also able to study separately our experience on women reporting a history of pleurisy. Unfortunately, the number able to give us details as to type was very small and our discussion is, therefore, limited to cases of unspecified type. These cases number 904, yielding an exposed to risk of 6,276 years of life. There were 20 deaths in the group during the period surveyed.

The mortality of these women has been especially favorable. On the basis of the American Men Table the actual mortality has been but 56 per cent. of the expected. The mortality has been better in the later insurance years than in those immediately

following issue, but in both cases it has been considerably less than that expected by the American Men Table. In the first five years it was 71.6 per cent. of the table and in the sixth and later years only 41.1 per cent.

Younger women, namely, those under attained age 35, suffered a higher mortality than those over age 35. The ratios of actual to expected deaths by the table were 73.3 per cent. and 46.4 per cent., respectively.

These exceptionally favorable mortality results among females reporting pleurisy have been due to the low rates among those whose illness occurred more than five years before issue. Those ill within five years of application for insurance showed higher mortality than the group as a whole regardless of the duration of the disease. In this group those reporting illnesses less than three weeks experienced a mortality 109.1 per cent. of the expected and those with longer illnesses suffered a slightly higher mortality. Unfortunately, the group is too small to make the actual size of these ratios significant, but the effect of the recency of illness is certain.

TABLE VII.

Mortality of Risks with a History of Pleurisy, Kind Not Stated

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Females. Classified by Time of Attack before Issue, Duration of Attack, and by Broad Attained Age Groups.

Attained age Time before issue Duration of attack	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Total	6275.92	20	56.8	51.9	71.6	41.1
Under age 35	3482.43	10	73.3	66.0	86.8	45.1
Ages 35 and over	2793.49	10	46.4	42.7	56.0	39.6
Less than five years						
Less than 3 weeks	1682.02	9	109.1	99.7		
3 weeks and more	692.15	4	125.4	115.3		
Five years and more						
Less than 3 weeks	2445.21	3	19.8	18.1		
3 weeks and more	971.24	2	35.1	32.2		

Of the 20 deaths among women with a history of pleurisy, seven were from tuberculosis. Four of these deaths occurred in the first five years after issue. The insurance had been issued within two years of the pleurisy attack in four instances.

3. Mortality in the Three Classes of Risks with Pleurisy Compared

At this point let us make a brief comparison of the experience in each of the three types of pleurisy which we have distinguished. In the aggregate, the mortality of risks with a history of dry pleurisy has been the highest of the three in the Metropolitan experience. The actual deaths in this group were 134.1 per cent. of the expected, as against 124.6 per cent. in the case of pleurisy with effusion, and 100.8 per cent. in the case of those giving no details regarding the type of pleurisy. The small difference amounting to 10 per cent. between the mortality of the dry and "wet" cases, we are inclined to dismiss as not significant in view of the relatively small size of the two groups. The favorable results on the "wet" cases undoubtedly reflect the very cautious attitude toward them on the part of the Medical Directors of the Company. The larger difference in mortality between the two groups specific as to type and the group without details is significant, but is undoubtedly due to the large but indeterminable proportion of such cases where the diagnosis was incorrect and the actual illness, often self-diagnosed, was merely a pleurodynia, an intercostal neuralgia, a chest pain or the like.

At ages under 35, the mortality in the group with effusion was only 83.2 per cent. of the expected by the American Men Table, but the group was too small for this result to be truly reliable. In the case of the dry pleurisies, the mortality was 158.7 per cent. of the table, and in the case of pleurisies of unspecified type 109.1 per cent.

At ages beyond 35, the cases with effusion suffered the highest mortality—151.5 per cent.; those with dry pleurisy, next highest —121.0 per cent.; and those with pleurisy of unspecified type, the lowest—97.6 per cent. In all three groups, the highest ratios of

TABLE VIII.

Mortality of Risks with a History of Pleurisy

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Classified by Type of Pleurisy and by Broad Attained Age Groups.

Attained age Type of Pleurisy	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
All ages						
Pleurisy with effusion	2821.17	19	124.6	113.2	142.1	106.5
Dry pleurisy	5122.58	39	134.1	121.3	154.4	110.7
Pleurisy, kind not stated	29602.14	191	100.8	92.4	110.9	91.4
Under age 35						
Pleurisy with effusion	1535.66	5	83.2	74.5	73.3	104.2
Dry pleurisy	2607.59	16	158.7	140.8	163.5	146.0
Pleurisy, kind not stated	13284.01	57	109.1	98.3	105.5	116.6
Ages 35 and over						
Pleurisy with effusion	1285.51	14	151.5	139.0	219.2	107.3
Dry pleurisy	2514.99	23	121.0	110.7	146.3	101.8
Pleurisy, kind not stated	16318.13	134	97.6	90.1	114.3	86.1

actual to expected deaths were found in the early years after issue.

4. Comparison of Mortality According to Standard or Substandard Ratings

It will be interesting at this point to review the actual results we have obtained in rating cases reporting a history of pleurisy. As we have stated above, we have combined in this report the issues on both standard and substandard policies on the assumption that the various factors entering into the rating of these applicants would tend to separate out the substandard issues from the rest. Most of these applicants were granted standard insurance which, with us, represents a mortality up to 125 per cent. of normal; a smaller number, Intermediate, which allows for an

extra mortality of 25 to 50 per cent. of normal; and a still smaller, Special Class, which permits from 50 to 100 per cent. excess mortality.

In the case of pleurisy with effusion and of pleurisy without details as to type, our experience shows good selection of the risks in that the mortality of those limited to Intermediate much exceeds that of those granted standard insurance, and the mortality of those limited to Special Class exceeds that of the Intermediate group. The differences roughly correspond to those expected. In dry pleurisy, however, the results of our rating have not been so very satisfactory. The mortality on cases taken for standard plans has been in excess of that permissible and not much lower than for those limited to Intermediate.

TABLE IX.

Mortality of Risks with a History of Pleurisy

Ratio of Actual to Expected Deaths by the American Men Mortality Table. White Males. Cases Rated as Standard and Substandard Separately. By Type of Pleurisy.

Type of Pleurisy Department of Issue	Years of life exposed to risk	Actual deaths	Per Cent. Actual of Expected Deaths			
			Aggregate		First 5 years	6th and later years
			Select and Ultimate	Ultimate	Select	Ultimate
Pleurisy with effusion....	2821.17	19	124.6	113.2	142.1	106.5
Standard	1885.62	10	90.7	83.0	100.0	82.9
Substandard						
Intermediate	454.57	3	143.5	126.6		
Special Class	485.02	6	281.7	255.3		
Dry pleurisy	5122.58	39	134.1	121.3	154.4	110.7
Standard	4179.66	33	135.1	123.2	155.5	114.8
Substandard						
Intermediate	613.18	5	160.8	138.9		
Special Class	329.91	1	64.9	58.1		
Pleurisy, kind not stated	29602.14	191	100.8	92.4	110.9	91.4
Standard	26326.99	161	95.0	87.1	103.6	87.7
Substandard						
Intermediate	2510.54	22	143.3	125.6	130.5	167.0
Special Class	764.57	8	196.6	178.2	315.3	54.1

Summary and Conclusions

We may now summarize the results of our study.

1. The mortality of all risks with a history of dry or wet pleurisy insured by the Metropolitan has been from 25 to 35 per cent. in excess of that expected by the American Men Table. In the large group of cases giving no details as to type the mortality was approximately equal to that table.

2. In all three groups, the mortality varied inversely with the length of the period since the illness.

3. The excess mortality was for the most part concentrated in the years immediately following the issue of insurance. Almost uniformly, the ratios of actual to expected deaths were higher in the first five years after issue than in the sixth and later years.

4. The mortality ratios on the younger applicants were higher than those on older ones.

5. Length of illness, i.e., whether illness was under or over two weeks, influenced the mortality of risks with a pleurisy history of unspecified type, the mortality increasing with the duration of disability. In dry pleurisy, this factor was of less influence. In these cases, the recency of illness was the more important factor.

6. Young underweights with a history of pleurisy presented a greater risk than persons of average weight. The factor of underweight is less important among older risks.

7. The excess mortality in pleurisy is largely due to subsequent tuberculosis.

8. The experience on female lives with a history of pleurisy has been better than that on males.

9. The rating of pleurisy cases, on the whole, has been successful in placing the risks in those classes which covered the extra risk involved.

By and large, we have in the past, been somewhat too generous with risks giving a history of pleurisy. We have especially underrated the seriousness of dry pleurisy. These statements apply

not only to our Company but to the insurance business as a whole, if we may accept the evidence offered by the Medical Impairment Study. In that report, you will recall that the experience on standard issues in all types of pleurisy was in excess of the expected. In the relatively large experience on dry pleurisy the ratio of actual to expected deaths by the Basic Table was 142 per cent. and on the smaller experience with wet pleurisy, 139 per cent.

It is not our opinion that all risks with a history of pleurisy must be limited to substandard insurance, but that they may be selected more successfully than in the past. No applicant with a wet or dry pleurisy where we are reasonably sure of the diagnosis should be granted insurance within two years of his illness, and if under 35 years of age until three years have elapsed. Under this age, few cases may indeed be insured on standard plans until ten years have elapsed; consideration being given to the various factors which we have shown to be of most importance in the prognosis. At ages over 35, a few cases may be granted standard insurance after five years have elapsed.

When details are not available as to the type of pleurisy, we should be relatively severe with cases who report illnesses lasting two weeks or more. Young persons in this category are not usually insurable on any plan until two years have elapsed, and if underweight, three years. Standard insurance may be granted as a rule on young persons of good build after five years, and among underweights who are otherwise unexceptional risks, after ten years. With older persons we can be somewhat more liberal. Carefully selected cases may be accepted within two years, but only the exceptional case at standard rates until five years have elapsed. After that, standard insurance may be granted more freely on these older risks, except those who are appreciably underweight, if there are no contra-indications, such as the occupation or the family history of the applicant. Those reporting histories of pleurisy of unspecified type lasting only a few days can be granted more generous treatment; indeed, in the majority of cases such a history is unimportant and may be disregarded.

As in the past due weight should be given to factors not dealt with in this paper, namely, the occupation of the applicant, his family history, the personal record of other illness besides the pleurisy, and the condition of the lungs at examination.

DR. CHRISTIERNIN—The discussion of this paper will be opened by Dr. William B. Bartlett of the John Hancock Mutual Life Insurance Company of Boston.

DR. BARTLETT—Mr. President and Gentlemen of the Association: I realize that a statistical paper of this nature lends itself to discussion about as readily as the multiplication table. If it wasn't for the fact that your President is not only my college classmate but my medical classmate, I shouldn't be here, but I am really very glad to have this opportunity, because I have had a chance to read Dr. Dublin's paper before the rest of you have had a chance to read it, digested it quite thoroughly, or as thoroughly as my digestion allows and I find there is a great deal in it. I think it is a poor idea to introduce the discussion with an apology, but before I get through you will see that the discussion is in no way as statistical as the paper.

As it is quite impossible to add anything of a statistical nature to an investigation of this sort, it occurs to me that it may be of interest to discuss the question of pleurisy from the clinical standpoint that we may ascertain how clinical experience agrees with the facts revealed in Dr. Dublin's study.

Dry pleurisy, pleurisy with effusion and empyema are stages in the same pathological process; the form of pleurisy depending on the nature of the infection and the resistance of the organism. It is helpful to remember that infection with the tubercle bacillus usually produces a clear fluid or a deposit of fibrin while the pneumococcus produces turbid fluid or forms pus. Empyema is almost always secondary, the pneumococcus and streptococcus being the usual causative agent and usually follows pneumonia, lung abscess or trauma. Its significance from an insurance standpoint is that of the causative agent. Cancer or sarcoma of the lung should be considered whenever a bloody fluid is

found. Recent investigations suggest that this form of cancer is more common than has previously been supposed. Because of its long duration and the absence of prominent symptoms in the early stages it may be of considerable importance from an insurance standpoint.

Pleurisies may be divided into two groups: 1. The so-called idiopathic pleurisy which comes on in a previously healthy individual without apparent cause and is usually attributed to catching cold. 2. Pleurisy secondary to some condition already recognized such as pneumonia, infarct, pulmonary tuberculosis, trauma, or a generalized infection. Secondary pleurisy need concern us no further as its importance from an insurance standpoint is entirely that of the original disease.

As Dr. Dublin states in his opening sentence, pleurisy is seldom a primary disease and the term idiopathic pleurisy is therefore a misnomer. It is convenient however to class all pleurisies, the etiology of which is not apparent, in this group. Their significance from the insurance standpoint is dependant on the fact that the causative factor is often the tubercle bacillus. This fact is well brought out by Dr. Dublin's figures; he finds the mortality greatest at the younger ages, in those of under-average build and to decrease with the number of years elapsed since the attack—all facts consistant with tuberculosis.

The results in pleurisy with effusion are just what we should expect from clinical experience. Lord claims that three-fourths of these cases are tuberculosis and other writers believe that the per cent. is even higher. Dr. Gerald Webb of Colorado Springs says "The general optimism regarding the outcome of pleurisy with effusion is not justified and pleuritic disease should be regarded as seriously as pulmonary hemorrhage". It is therefore not surprising that the death rate from tuberculosis is higher than normal. In the recent Medical Impairment Study it was six times the normal. Thus clinical experience and statistical study both prove that pleurisy with effusion is a serious impairment and it should be so considered. I believe, however, that the method of treating this impairment suggested in the new Medical Impairment Ratings is unnecessarily severe. Pleurisy

with effusion is there rated the same as pulmonary tuberculosis. Dr. Dublin's figures do not suggest this degree of severity nor does clinical experience. If seventy-five per cent. of these cases are tuberculous a rating of two-thirds or three-quarters of that for pulmonary tuberculosis would seem more reasonable.

Fulton and Hahn recently investigated the subsequent history of forty patients treated for dry pleurisy at the Peter Bent Brigham Hospital. These patients were followed from one to sixteen years (average seven years). Only four developed pulmonary tuberculosis. They conclude that while one should appreciate the likelihood that an acute dry pleurisy may be an early manifestation of tuberculosis infection, pulmonary tuberculosis develops in fewer of the cases than has been taught previously.

Notwithstanding this optimistic report we must remember that most clinical observers believe that wet and dry pleurisy are merely different stages in the same process and have a similar etiology. The two forms represent either a difference in the severity or extent of the infection or are an expression of the resistance and reaction of the individual. Acute dry pleurisy frequently develops into pleurisy with effusion, a fact that suggests it is but a mild or early expression of a pathological process that may result in the formation of fluid. If such is the case one may look on an acute dry pleurisy as just as good evidence of tuberculous infection as is pleurisy with effusion. Such is the tendency in the modern text books. Lord followed sixty cases of dry pleurisy and found 30% developed tuberculosis. He believes that dry pleurisy is as liable to be followed by tuberculosis as wet pleurisy. Von Ruck shares this belief. The unfavorable experience of the Metropolitan with cases of dry pleurisy is therefore not surprising. Since pleurisy with effusion has been usually considered the more serious impairment the selection has been stricter when this was recorded and a better mortality has resulted.

An interesting point brought out in this paper is that the mortality varies directly with the duration of the attack. The shorter the attack the lower the mortality. This is undoubtedly

a reflection of the fact that many of the short illnesses called pleurisy are in reality due to other less serious causes. There are probably few diseases more often diagnosed without adequate reason than pleurisy. Many times a simple attack of muscular rheumatism, muscle strain or intercostal neuralgia is so called. This diagnosis is remembered long after the symptoms and duration of the illness are forgotten. These cases help to improve the mortality in the group of short duration. The favorable mortality noted by Dr. Dublin in the group with a family history of tuberculosis suggests the possibility of an acquired immunity from childhood infection.

In conclusion let me emphasize the need for a careful history and a thorough examination of all applicants with a history of pleurisy. The examination will exclude those who have developed pulmonary complications or extensive adhesions; the history will exclude those that have been erroneously diagnosed. If the cases are carefully selected the experience will be satisfactory. Unfavorable factors are youth, light weight and a history of a recent or prolonged attack. Favorable factors are weight above the average, a brief and remote attack.

Dr. Dingman—Doctor Dublin and Mr. Marks have utilized valuable data in the Metropolitan files to prepare for us a paper that maintains the usual high standards of the authors.

Pleurisy, which should really be called pleuritis, is an inflammation of the double fold membrane that encloses the lung. Inflammation occasions effusion of lymph, but the exudate may be so little that the pleurisy is said to be dry. When the effusion is appreciable, the pleurisy is said to be wet. And if the serous exudate becomes purulent, empyema is the term. Inasmuch as the space between the two-ply pleural membrane is a closed cavity, a pleural invasion is necessarily secondary to an infection elsewhere in the body. Nevertheless, the elsewhere source is not always demonstrable and some pleurisies appear to be primary. Incorrect though the term may be pathologically, it conveys an exact idea when, clinically or insurancely, we speak of pleurisy as being primary.

Primary pleurisy always suggests tuberculosis. Insurance thought is in accord with clinical that pleural inflammation without a definitely recognized primary infection must be considered tuberculous. Medical directors appreciate this principle and the severe selection standards applied to histories of primary pleurisy have done much to bring about a mortality experience more favorable than might ordinarily be expected.

What further saves the experience is the inclusion of risks whose pleurisies are secondary to infections such as pneumonia and influenza, and the inclusion of pleurisy cases that are not pleurisy at all. Pleurisy is one of the most frequently mislabeled of diseases, and many are the stitches in the side that are merely neuralgic or myalgic in nature, that cause no disability nor even a fever, but nevertheless acquire the pleurisy label. They shorten longevity not a whit, but they do seep in and dilute our classes of true pleurisy.

The excess mortality found so uniformly in the Metropolitan experience when the pleurisy history was recent may have its explanation in part, at least, because applicant and examiner and family doctor are close enough to the facts that pleurisy so-called is truly pleurisy. Pleurisy more than five years previous to application is not so subject to corroboration and the group contains more falsely labelled cases.

It is doubtful if underwriters should make differentiation between dry and wet pleurisy. Dry pleurisy is not dry at all except comparatively; it is wet, but just a little bit wet. Wet pleurisy is a later stage. The advantage of the terminology is the inference it permits that the diagnosis has been carefully made. A more practicable classification for selectors of risks is according to length of illness, as the authors have well shown. If long periods of disability do not prove the diagnosis, they do at least exclude the inconsequentials and they do indicate inadequate resistance to the ailment. The excess mortality is due chiefly to tuberculosis which hazard is greatest, always, in underweights at the younger ages.

The Association is indebted to Doctor Dublin and Mr. Marks for so splendid a paper from the Metropolitan files.

Dr. Dublin—Mr. Chairman, I have no further comment. It seems to me that Dr. Bartlett has fully supplemented the statistical paper with a very clear medical insight and the two points of view combined give you a really good working basis. Perhaps my colleague, Mr. Marks, who really carried the brunt of this study, may have a few comments.

Mr. Marks—About the only comment I would have to make is regarding the study recently made in the middle west at the Glen Lakes Sanatorium in Wisconsin and they found out there that in their histories on tuberculosis, by far the larger number of them gave histories of dry pleurisy rather than wet pleurisy.

Dr. Christiernin—The next speaker requires no introduction to an audience of physicians. Dr. Frank H. Lahey of the Lahey Clinic of Boston will speak to us on "Thyroid Disease".

THYROID DISEASE

By Dr. Frank H. Lahey
of the Lahey Clinic of Boston.

Mr. President and Members of the Association of Life Insurance Medical Directors of America: I presume our viewpoint on thyroid conditions may be quite different from yours. I would like, however, to present to you the results of our experience along the lines which might be of the greatest value to you. I have no doubt that because I have little experience with life insurance affairs, I will not do it very satisfactorily. I am much more used to presenting the subject from the point of view of: What is the matter with them? What happens to them immediately? What is the immediate mortality? and What is the ultimate outcome as far as cure goes? Therefore, I have selected from our experience a set of slides illustrating certain of the aspects of thyroid conditions and I will try to speak about them as much from your point of view as possible. Undoubtedly, as I say, however, I will not do it very satisfactorily. The slides probably will run over the time which is allotted to me, so if Dr. Christiernin will speak to me, I can stop at any time and I wish he would allow enough time so that if you feel disposed, you may be able to ask me any questions you wish from a life insurance examiner's point of view about what happens to thyroids. Perhaps I will have to tell you that I do not know in many cases, but if I am able to answer any of the questions about things you would like to know about thyroids, I will gladly do it.

Now I feel a little self-conscious to relate numbers to you, but I think one must qualify his statements on the basis of experience. I told Dr. Christiernin last night at the dinner that I expected that by the time I arrived home we would have operated on twelve thousand goitres. Therefore, we have had a real opportunity to know something about what happens to the

various types of goitres. You would be interested, I think, to know what the percentage group of the various types is in our experience. In New England—where thyroid conditions, of course, are quite a little different than in regions where goitre is endemic—we have averaged about fifty per cent. exophthalmic goitres, primary hyperthyroidism. We have averaged about twenty-six per cent. toxic adenomata. Therefore, we have run quite regularly three-fourths toxic cases and about one-fourth adenomata, cysts, and various other types in the form of malignant goitre.

One of your members requested me to report to you our end results on the so-called thyrocardiacs and I cannot do it because we reported our end result studies on the thyrocardiacs about five years ago, and while all the letters asking the patients to come in to bring the series up to date were sent out, and half of the patients have come in for follow-up examination, there still remain another half to be examined before we can report on end results. I can, however, state to you our convictions as to the effect of hyperthyroidism on the heart. I think one can say—I have just come from the American College of Surgeons meeting in St. Louis and the International Post-Graduate Assembly, where there have been papers on the thyroid heart (some of which I do not agree with)—that there is no actual effect by hyperthyroidism on the heart. I think one can say that prolonged hyperthyroidism produces no real heart damage. There is no evidence to prove that prolonged hyperthyroidism produces heart damage. In fact, there is every evidence to prove that it is not so. We feel that when a patient is relieved of thyroid toxicity, his heart is just as good as it ever was.

Now why do we believe this to be true? We have seen eighty-five patients die with goitre. We have seen a number of young people who have had hyperthyroidism over a long period of time die. When they die, they do so with uncountable pulse rates, extremely toxic; they often have had intense intoxication over a considerable period of time, but provided they do not have heart damage, due to causes other than hyperthyroidism, they practically never show cardiac decompensation. They were able

to lie down. They do not have enlarged neck veins. They do not have enlarged livers. They do not have anasarca, and at autopsy they show no pathological evidence of heart damage. There have been a few experiments conducted to show heart damage, by feeding thyroid extract to animals, but there is no convincing evidence that the effect of increased thyroid secretion produces heart damage. There is not a true thyroid heart, but there is heart failure associated with hyperthyroidism, but it occurs almost entirely in older people who have lived long enough to have heart damage from other causes.

The effect of hyperthyroidism in producing heart failure and heart irregularity is on a previously damaged heart. Given a patient with heart damage of any type, then the overburden and the overdrive and the increased heart metabolism produce first the irregularities of a transient character, then of a fixed character and finally failures, and while subtotal thyroidectomy by the removal of the hyperthyroidism can overcome the decompensation, it cannot, of course, make over the damaged heart. Therefore, one may use the fact that a superimposed hyperthyroidism produces heart failure as quite positive evidence that if the heart fails with superimposed hyperthyroidism, there is almost of necessity heart damage. We have learned certain things regarding irregularities in relation to hyperthyroidism. Given any individual with an unexplained auricular fibrillation, one must rule out hyperthyroidism. Hyperthyroidism produces more unexplained auricular fibrillation, we believe, than any one other condition. As to auricular fibrillation and hyperthyroidism, one can be quite sure that the patient with transient auricular fibrillation and hyperthyroidism will not fibrillate again as far as his immediate disease goes if his hyperthyroidism be removed. On the other hand, if he has an established auricular fibrillation, but a small percentage of those cases will have their regular rate restored by the removal of the hyperthyroidism. However, after the removal of the hyperthyroidism, over fifty per cent. of these cases can be restored to regular rhythm by quinidine. One should not give quinidine to patients who have hyperthyroidism, preoperatively—and I say this in a general way

because it may not interest you from the point of view of life insurance examiners, but it is a good point to know. One should not give quinidine to patients with toxic goitre preoperatively for two reasons: One, because they do not stand quinidine as well as patients unaffected by hyperthyroidism; two, because the quinidine is wasted.

If you give patients with hyperthyroidism quinidine, you can restore their absolute irregularity to normal rhythm, but when they are operated upon, they will go into abnormal rhythm again as a result of the post-operative reaction, and therefore you have wasted the quinidine. Furthermore, because of the idiosyncrasies of patients to quinidine, occasionally quinidine in a patient with hyperthyroidism may result in a serious outcome. We much prefer to use digitalis preoperatively and quinidine postoperatively. With digitalis you cut down his auricular-ventricular disproportion and he is definitely a better operative risk. Then remove the toxicity by subtotal thyroidectomy and if there is still irregularity, you can give quinidine safely.

I would like to show you some slides, perhaps illustrating points which may be of some value. May I have the first slide, please?

We have been very much interested in the group of patients who have low basal metabolic rates but who have no clinical evidences of myxœdema. Dr. L. M. Hurxthal, working in the Medical Department of the Clinic, has demonstrated a quite valuable method of segregating those patients with low basal rates who have true myxœdema from those patients with low basal rates who have no myxœdema. In segregating those patients who will be benefited by thyroid extract from those who will not be benefited by thyroid extract, based now on our experience with low basal rates, we have never but once seen a patient with a metabolism of lower than minus twenty-four who did not have clinical evidences of myxœdema, so that you and I could make the clinical diagnosis. On the other hand, we have seen a large number of patients who have minus ten, minus fifteen, minus eighteen, nineteen, and twenty, and so on, who have been

on thyroid extract feeding for long periods of time and who actually are not benefited by the thyroid extract whatever.

Dr. Hurxthal is now publishing in sections the result of several hundred blood cholesterols done throughout the past year or year and a half, so that these are but the preliminary figures, but on the other hand, they run very parallel to the larger group which is now being published. If the normal blood cholesterol be approximately two hundred milligrams, a hypercholesterolemia will be found in true myxœdema averaging in a small series of twenty-three cases three hundred milligrams and ranging as high as five hundred milligrams. When these patients are placed upon thyroid extract and the basal metabolism comes to normal, hand in hand with their drop in basal rate goes the drop in blood cholesterol to normal.

In those patients with minus degrees of basal metabolism but without clinical evidence of myxœdema, there will be a normal or low blood cholesterol when the basal rates are not due to diminished thyroid function and in such cases the feeding of thyroid extract will do no good. The blood cholesterol serves to distinguish the group which has myxœdema from the group which does not have myxœdema. It does not do any good to give that group of cases thyroid extract and one may say, I think, with quite reasonable safety that even though they have low metabolisms, they do not have true myxœdema. It is a fact that the range of normal metabolism is not fully understood and there seems little question but that it is normal for some individuals to have relatively low metabolisms.

Now, we have been interested also in what happens to cardiac patients who have myxœdema. We have proven to our satisfaction now by operating upon several hundred thyroid cardiacs that when a patient has heart failure and superimposed hyperthyroidism, if you do away with the hyperthyroidism by partial thyroidectomy, they lose their failure and regain a marked degree of cardiac capacity. So we have likewise learned that these patients who have cardiac failure with associated myxœdema do just the same thing. When you restore their basal metabolism to normal with no digitalis, no cardiac treat-

ment, only with thyroid extract, then you also decrease the size of their heart and restore their cardiac compensation, just as you do in the hyperthyroidism cases with associated heart failure. Here is a case of complete myxœdema, large heart, and fluid associated with it.

Here is the same patient who has been only put to bed and put on thyroid extract and nothing else done, yet she has completely regained her compensation. One should have in mind, therefore, that the effect of myxœdema is very similar on damaged hearts to that of hyperthyroidism and that they can be relieved in the same way by the removal of their hypothyroidism.

What happens to children who have exophthalmic goitre? This is a little girl three and a half years old. This is a typical case of exophthalmic goitre. We have now operated on a large number of children with exophthalmic goitre, ranging in age from three upward, and I think one can say that they do just the same as adults do. We were very much disturbed in the beginning of our experiences with exophthalmic goitres in children for two reasons: We did not know what their immediate operative reaction would be because of children's ability to react to a trauma or intoxication to such a great degree. The mortality in children, however, is no higher than in adults. The other thing that interested us was, How much danger is there of myxœdema in children? There is a good deal. A child needs his thyroid very much more than an adult. Therefore, I think, from an insurance point of view, I would be a little more uncertain about a child who had an exophthalmic goitre operated upon than I would about an adult, because thyroid extract serves very well as a substitute for thyroid, provided one has attained full growth and mental development. On the other hand, the thyroid gland is not only essential in the control of heat production, but it also has a growth element, as proven by the presence of cretinism so much in Switzerland. There are a certain number of children, I believe, who will get myxœdema. Therefore, from an insurance point of view, it would seem to me that a child who had a subtotal thyroidectomy would be a little

higher risk than an adult. You can get rid of hyperthyroidism, but if myxœdema intervenes, you may have difficulty with growth development.

This is the type of hyperthyroidism, apathetic hyperthyroidism, which I think should interest insurance examiners. We have described this type and repeatedly written about it. This is the type which escapes detection so easily. Everyone is familiar with the type as seen in that little child with a typical exophthalmic goitre, because she is the typical text-book picture of exophthalmic goitre. She flies all the danger signals of the disease. She has stare, exophthalmos, tachycardia, thrusting apex beat, toxic pulse rate, hot skin, tremulosity, irritability, everything that goes with it. This lady with apathetic hyperthyroidism has everything that the child has, only in a reversed manner. This is the type of hyperthyroidism we have described as apathetic hyperthyroidism. They are one and the same disease, I believe, only they are different reactions of different organisms to it. The young resilient individual reacts with activation, the aged individual, often with long-standing hyperthyroidism, reacts in a much more sluggish manner to it. In this type of apathetic hyperthyroidism one does not see eye signs as a rule, almost never exophthalmos, rarely even stare. One does not see the flushed skin. The skin is usually markedly pigmented. When they are operated on and this hyperthyroidism is removed, the pigmentation disappears. We have taken sections of the skin repeatedly to try and see what the pigmentation is, but we cannot find out. They have cool wrinkled skins. They have relatively slow pulse rates—I mean around one hundred to one hundred ten. They have a firm thyroid gland. They do not have marked elevation of basal rates. They have, however, had progressive weight loss over a long period of time, sometimes up to one hundred pounds. They have marked myasthenia. They are, I believe, the most dangerous type of hyperthyroidism from the point of view of operability and also the ones most often overlooked. What should make one suspect the presence of the so-called apathetic hyperthyroidism is the particularly slow and progressive unexplained weight loss, the

moderate but persistent elevations of pulse rates and also the fact that they run moderately but persistently elevated basal rates. Now, to prove that this is quite a different condition, perhaps the same in origin but a different state from the little girl that you saw with activated hyperthyroidism, if they die, they die quite differently. The patient who has true activated exophthalmic goitre dies actively motorized. The patient with apathetic hyperthyroidism dies in a coma. That little girl, if she died, would thrash around in bed and would require forcible restraint to keep her quiet; her pulse would be at an uncountable rate. If this patient with apathetic hyperthyroidism died, she would die by going back to bed after an operation, go quietly to sleep and never wake up. So that is the type which we have described as apathetic hyperthyroidism so readily overlooked. The results in these cases are excellent as far as the removal of the thyroid goes in ultimate outcome. They gain weight and we get, I believe, just as good postoperative results as with patients with activated hyperthyroidism except that they, of course, are old individuals and they do not possess the capacity to regain the degree of health that the younger individuals do.

What should one expect regarding the eyes in exophthalmic goitre, primary hyperthyroidism? Everyone who has exophthalmic goitre becomes a candidate for blindness. We have seen six individuals lose their eyes from exophthalmos. This should never happen today. I show you this slide of a patient losing her eyes, not because it has any particular relation to life insurance, but one should always have in mind what can happen to patients with exophthalmic goitre. We have tried everything to relieve this excessive exophthalmos, but there is only one operation that will relieve these patients and that is the operation devised by Dr. Howard Naffziger of San Francisco, and that is a cerebral operation in which the front of the scalp is turned down, the frontal lobes are lifted up, and the whole orbit with the Ring of Zinn is decompressed. What these patients suffer from is œdema of the muscles behind the orbit and they have to have a decompression just the way a brain

tumor does and then their eyes can be saved. One should realize that many of these very bad cases of exophthalmos have come in the presence of myxœdema after the removal of the thyroid. These patients should be submitted immediately to this cerebral operation which will save their eyes and should not be tinkered with over a long period of time, because the less radical measures will not save their eyes.

What is the effect of iodine on exophthalmic goitre? You, from a life insurance examiner's point of view, will be interested to know whether or not iodine in itself accomplishes cures. I do not say this with bias because we run a clinic where thyroid conditions are handled in large numbers—but, as a life insurance examiner, I would be very much interested in determining what the chances of recurrence after thyroidectomy were going to be. In this connection, I would be interested in who did the operation, not the individual but where, because success in thyroid surgery is related so much to how much thyroid tissue is removed, and the variation in type of thyroid tissue when it is visualized at operation together with the reaction to iodine settles the amount of tissue to remove.

(Slides showing effect of different amounts of iodine in the tissues of patients with exophthalmic goitre were presented.)

Dr. R. B. Cattell in our Clinic has reduced four hundred surgically removed glands to a powder after having made histological sections. On these he did the milligrams of iodine per gram of dried gland and one finds that with iodine involution (the change in the histologic picture wrought by iodine) a clinical improvement goes hand in hand with the increase in milligrams of iodine in the thyroid. On the other hand, one should realize that while this involution in the gland shows evidence of diminished activity—and this histologic picture which looks like inactivity—is somewhat misleading, the histologic picture of the apparently inactive gland does not go hand in hand with the ability of the part left behind to function. The patient, a section of whose gland shows marked involution, still has a

metabolism of forty-six and a high pulse rate, and was finally relieved only by subtotal thyroidectomy. While iodine will produce temporary improvement if the patient is continued on iodine over a long period of time, the basal metabolism returns to what it was originally or goes higher. Therefore, iodine is of only temporary value as a pre-operative method of preparation.

What is the result of subtotal thyroidectomy on blood pressure? We have been somewhat misled, I feel quite sure from our experience, by statements which suggest that if you remove thyroid toxicity, you decrease hypertension. That is not a fact, based on our experience, and it is of interest to you to know what is not so as well as what is so. For instance, we have charted the basal systolic blood pressure in non-toxic goitre before operation and exophthalmic goitre six to twelve months after operation. They run parallel courses. The increase in pressure goes up as they progress into the advanced age groups, but otherwise the post-operative blood pressures run parallel courses in toxic and non-toxic goitre patients. The parallel course is similar in exophthalmic goitre patients six to twelve months after operation as compared with office patients and toxic adenomatous goitre three months after operation. There is no particular difference between a group of patients who come to the clinic just for general examinations and those after operation who have had exophthalmic goitre or toxic adenomas. They also increase as they go into the advanced age groups.

We have compared in the same way the postoperative blood pressures several months after operation in patients with severe exophthalmic goitres all done in two stages. They had to be severe hyperthyroidism to be operated on in two stages. In this group we took the average basal blood pressure before and after operation in different age groups in one hundred cases of very toxic goitre, all operated because of the seriousness of the disease in two stages. The outstanding feature which occurs in toxic goitre is an increase in pulse pressure. Following relief from toxicity, you can count on a slight diminution in the pulse blood pressure, but you can not count on a diminution in the blood pressure picture. You see a small but definite drop

in the pulse pressure but there is no particular effect upon the blood pressure itself, the trend being generally upward with years and paralleling the pre-operative rate.

I would like to call your attention now, from the point of view of risks, to the group of adenomatous goitre. How dangerous are these adenomata of the thyroid? I think this question must be difficult from a life insurance examiner's point of view, but I can point out to you which one is dangerous and why. Let us take the single, discrete adenoma of the thyroid, for example, about the size of an egg. What is dangerous about that?

This is what is dangerous about that: Vessel ingrowth. We did not use to know this miscroscopic and pathological feature about thyroid adenomata. If one will search beneath the capsule of seemingly benign thyroid adenomata and not infrequently we find places where tissue apparently benign in character has penetrated and invaded the blood vessel wall. These are the so-called malignant adenomata of the continental pathologists. We have found that six per cent. of all the patients with seemingly benign, discrete adenomata of the thyroid show blood vessel invasion. Not every patient who has blood vessel invasion has cancer, but most patients who do have a carcinomatous adenoma show blood vessel invasion. The thing we cannot tell you and no one can tell you is what number of these patients with thyroid adenomata showing blood vessel invasion are going to have later malignancy. We can tell you this, that ninety-six per cent. of all the cancer of the thyroid which we see arises in the previously existing benign thyroid adenomata, that one in sixteen of every one of the patients who have an adenoma benign in character will show blood vessel invasion, that two and eight-tenths per cent. of all the adenomata will show frank malignancy. Therefore, every patient who has a discrete tumor of the thyroid becomes at once a candidate for malignancy and should have his adenoma removed. We feel very strongly that if an adenoma is discrete, its capsule is unruptured, and it is removed, they are practically freed from the danger of malignancy and they have no more likelihood of having an adenoma

than you or I do. The patients who have discrete adenomata are no more prone to have other adenomata in our experience than the patient who starts originally without them.

There is a type of thyroid tumor (aberrant thyroids) which life insurance examiners should be on the lookout for. It is not common; it is easily diagnosed, once you are familiar with it, and it is potentially malignant. There are only a few cases reported in the literature. Dr. R. B. Cattell has published and described seventeen cases from our Clinic. One should be on the lookout for these, I think, and I will describe them to you. In most cases the thyroid originates at the base of the tongue and descends in the fœtus through the tongue to its place on the tracheal rings. There are, however, other origins. There are two lateral origins of the thyroid, occasionally from the ultimobranchial bodies and in certain instances these send in thyroid tissue from the side and produce lateral aberrant thyroid tissue. These, when exposed surgically, are bluish black in color. They are quite peculiar in appearance. They look almost like melanotic sarcomata, they are papilliferous in character, and tend to become malignant. What are their clinical features? When you find a patient with a chain of soft tumors, just like small lymph glands, running up and down in front of his sternomastoid muscle and along the course of the internal jugular vein on either side, movable, not tender, very much like lymph glands but not enlarging, they are in most cases lateral aberrant thyroid tissue. They consist of a papilliferous type of tissue which is quite apt to become malignant. However, like all papilliferous types of tissue, they are extremely radiosensitive. Two of these seventeen cases have died of malignancy; the others are well and alive for a varying number of years following radical removal and X-ray treatment.

You will be interested to know what our impressions and experiences are regarding intrathoracic goitre. How deep can it be? What kind of risks are they after they are out?

Once they are out and once the cavity has closed, I see no reason why these patients, even with very deep intrathoracic goitres should not be excellent risks from every point of view.

We have now operated on several hundred intrathoracic goitres, some at enormous depths and all have done well provided they have gotten through the operation, and there has been no damage done to any adjacent structures. I see no reason, from our experience, why they should not be just as well as they ever were.

What in a general examination should make you suspect the presence of intrathoracic goitre? These intrathoracic goitres can often exist deep in the mediastinum with nothing to show on the neck. Therefore, you should always be suspicious of the presence of dilated chest veins. Dilated superficial chest veins should make you suspect intrathoracic goitre and the presence of dilated chest veins should call for X-ray to make sure that they do not have intrathoracic goitre. When a large goitre is present within the mediastinum, it not infrequently causes some pressure upon the internal jugular veins interfering thus with the return of blood from the head and resulting in a compensatory dilatation of the superficial chest veins. In such cases X-rays of the upper chest will rarely fail to demonstrate the presence or absence of an intrathoracic goitre in the mediastinum.

How should we report end results in the treatment of hyperthyroidism, and then I am finished with these slides and if you like, and we have time, you may ask any questions you wish.

I meet around the country men who treat exophthalmic goitre by various methods, who go about presenting end results with pictures taken before and after treatment. That is quite wrong. Why? Because you and I have seen cures of exophthalmic goitre as a result of no treatment whatever. About one in every fourteen cases of exophthalmic goitre runs a self-limited course, and so that is the wrong way; end results must be presented in groups.

Here is a group of one hundred eighty-eight patients with exophthalmic goitre whose basal metabolism before operation average plus thirty-nine and whose basal metabolism after operation average minus three. We should report our end results by showing groups of metabolism averages before and a year after treatment. That even is not the right way to report final

end results. That proves that subtotal thyroidectomy in a group does produce good end results, but it is not specific about what happens in the group. We might have a large number of myxœdemas and a large number of persisting hyperthyroidism which balance each other and so make the figures come out that way.

We have been interested, therefore, in learning how many of these patients following exophthalmic goitre operations have myxœdema and how well they were some years after operation. Therefore, we did a five-year follow-up on ninety-seven cases. All had been operated upon five years previously. All were seen personally by Dr. H. M. Clute or Dr. Ross Veal five years after operation. They all had metabolisms and were not long-range follow-ups. The ninety-seven cases showed three cases of myxœdema. That is about the average percentage of myxœdema that one will obtain after subtotal thyroidectomy. I would like to say to you that in my opinion a patient with myxoedema is not quite the same when he is on thyroid extract feeding as he is when on his own thyroid. They are able to go on and be around, but I personally feel that myxœdema is undesirable, that any patient is a more normal individual in every way when his own thyroid is functioning.

Here are the end results of ninety-seven cases examined carefully at the end of five years. Absolutely well, eighty-two. I mean by that well from the point of view of a most critical analysis. Well after re-operation, seven. Well, but requiring thyroid extract, three. Total, ninety-two with operation, re-operation, and thyroid extract, or ninety-four per cent. are well.

What is the mortality from the point of view of the surgery? This, I think, has perhaps little or nothing to do with your experiences, but nevertheless is interesting. Now here are our mortality rates year by year. In the last six years for exophthalmic goitre we have been able to maintain a case mortality of seven-tenths of one per cent. and an operative mortality of fifty-two hundredths of one per cent. The risk of the operation in the hands of those who have had experience with it in large numbers is relatively slight.

DR. JIMENIS—Mr. President and Fellow Members: Dr. Lahey has not disappointed us. We expected something interesting and important and that is what he has given us. He has given us so much that I rather think that most of us will have to wait until the notes are transcribed and we can read it before we can get full benefit out of it.

I rather feel that he took an unfair advantage of me, because besides not giving me anything that I could study beforehand, he put the light out so that I couldn't make any notes. I shall not recite my own experiences in operating on goitres, because that would take us back before the War during my interne days. The results then were so poor that generally the removal of the offending organs was only the final resort and surgeons might be said to have finally removed the thyroid because there was nothing else left that he could do to it.

This approach forcing system in getting rid of the thyroid has now been displaced, excepting in a few cases where ligations still seem the wise course, by the really remarkable example set by men such as Dr. Lahey.

We have studied our own insurance experience on goitres, beginning with the issues of 1912 to 1928, carried to their anniversaries in 1931. This comprises 10,529 cases with an average age of approximately 27 years, and includes cases which showed a non-toxic goitre at the time of examination, and cases which gave a history of having had at some time in the past either non-toxic goitre or goitre with hyperthyroidism. Of course, these latter showed no signs of goitre or hyperthyroidsm, with the possible exception of exophthalmos, when they were insured. Our group includes both Standard and Substandard issues; but the goitre, or history of goitre, is the only impairment present.

I am going to show you the tables and let you judge for yourselves if we can learn anything from them. I think we can.

Here is our mortality by our own selection. This table and the next include all non-toxic goitres and those cases which

gave a history of toxic goitre at some time previous to their examination. In other words our entire group.

TABLE 1-A

Mortality of Males with Non-Toxic Goitre Present on Examination. Ratio of Actual to Expected Deaths by American Men Mortality Table.* By Department of Issue and by Broad Age Groups.

Department of Issue Attained Ages	Exposed to risk	Actual deaths	Per cent. Actual of Expected Deaths		
			All years	First 5 years	6th and later years
All cases					
All ages	15575.84	49	65.2	60.7	71.2
Under 35	11083.39	28	65.4	59.3	77.8
35 and over	4492.45	21	65.0	63.7	66.0
Ordinary (810 cases)					
All ages	4566.01	22	96.2	72.9	123.7
Under 35	3109.09	12	100.4	61.9	180.9
35 and over	1456.92	10	91.7	93.7	90.4
Intermediate (1,665 cases)					
All ages	9767.57	21	46.8	54.8	34.3
Under 35	7268.44	14	49.9	57.8	33.2
35 and over	2499.13	7	41.7	48.0	35.5
Special class (154 cases)					
All ages	1242.33	6	81.6	65.8	92.8

*Select and Ultimate Basis.

These are the males—They are are classified by the rating imposed at the time of issue into Ordinary, Intermediate and Special Class. The group is small and our judgment, while satisfactory on the whole, was not very accurate in each of the three subdivisions. This entire group starts with the issues of 1912 and our opinion concerning goitres has been considerably modified in that interval. In other words we have not imposed the same ratings on these goitres during this interval of 16 years and this may partly explain the result.

We did very well with the females, the mortality running along well within the limits we anticipated.

1-B

Mortality of Females with Non-Toxic Goitre Present on Examination. Ratio of Actual to Expected Deaths by American Men Mortality Table.* By Department of Issue and by Broad Age Groups.

Department of Issue Attained Ages	Exposed to risk	Actual deaths	Per cent. Actual of Expected Deaths		
			All years	First 5 years	6th and later years
All cases					
All ages	36334.03	156	87.3	92.8	80.0
Under 35	25560.60	87	88.5	97.1	70.2
35 and over	10773.43	69	85.9	84.7	86.9
Ordinary (1,852 cases)					
All ages	9957.99	31	68.9	73.4	62.0
Under 35	7563.24	22	76.5	73.0	85.4
35 and over	2394.75	9	55.4	74.7	41.9
Intermediate (3,935 cases)					
All ages	24161.58	102	87.5	97.3	73.4
Under 35	17032.96	58	88.3	103.9	56.2
35 and over	7128.62	44	86.5	85.4	87.5
Special class (278 cases)					
All ages	2214.42	23	135.6	128.4	139.8
Under 35	964.32	7	180.4	189.6	169.5
35 and over	1250.10	16	122.3	97.1	133.9

*Select and Ultimate Basis.

In this table, as in the previous table, you notice that in each age classification the younger ages fail to show a better mortality than the older ages.

As compared with the men, the women showed a higher mortality in the whole group, 87.3 vs. 65.2 for the men.

In the ordinary issues alone the men had the higher mortality, while in the substandard groups the women had the higher mortality.

These are comparisons of minor interest; but I do want you to note that in every case where the material was large enough to divide the younger ages they show a slightly higher mortality.

TABLE 2

Mortality of Females with Non-Toxic Goitre Present on Examination. Ratio of Actual to Expected Deaths by American Men Mortality Table.* Cases Classified According to Weight and Pulse Rate. By Broad Attained Age Groups. Standard and Sub-Standard Issues Combined.

Attained Ages	Exposed to risk	Actual deaths	Per cent. Actual of Expected Deaths		
			All years	First 5 years	6th and later years
All cases, total	36334.03	156	87.3	92.8	80.0
Ages under 35	25560.60	87	88.5	97.1	70.2
Ages 35 and over	10773.43	69	85.9	84.7	86.9
Cases with average weight and pulse below 80, total	12780.31	64	99.0	102.8	94.0
Ages under 35	8515.23	30	91.4	89.4	95.7
Ages 35 and over	4265.08	34	106.9	123.4	92.9
Cases with pulse rates 80-89, total	9625.74	32	69.5	76.4	59.4
Ages under 35	6979.89	19	71.5	84.1	39.6
Ages 35 and over	2645.85	13	66.9	59.0	73.1
Cases under average weight, total	19576.53	76	79.9	87.3	70.4
Ages under 35	14211.11	48	87.7	102.9	56.2
Ages 35 and over	5355.42	28	69.4	53.2	81.2
Cases 10%-15% under average weight, total	8288.15	33	81.2	91.3	68.0
Ages under 35	5817.21	19	84.4	103.8	42.3
Ages 35 and over	2470.94	14	77.1	65.9	85.2
Cases underweight with pulse rate 80-89, total	5648.55	16	59.1	69.0	44.9
Ages under 35	4155.63	10	63.2		
Ages 35 and over	1492.92	6	53.3		

*Select and Ultimate Basis.

These are our non-toxic goitres present on examination, female lives. Standard and Substandard issues are included together and they are subdivided according to the presence of slightly increased pulse and moderate underweight, these conditions in

themselves not being sufficiently marked to constitute a separate rateable impairment. The proportion of Standard to Substandard is about 4 to 9; in other words, a little less than one third of these were issued Standard insurance. This group is selected for these sub-classifications simply because it is the largest group we have.

Now I was strongly tempted not to show you this table because of the unexpected result in comparing these sub-classifications. They give us a better mortality when the goitre is accompanied by some underweight or slight increase in pulse rate and this conclusion, of course, has to be rejected outright. I don't know if the obvious explanation of careful selection will suffice after looking at these figures. Without doubt, everyone having anything to do with an application for insurance, which shows this combination, would not advise the issue of a policy unless in every other respect the risk appeared better than the average. What I get out of it is that slight variations in a pulse rate below 90 as reported to us cannot be important. Our examiners know well enough that 90 is considered our normal limit, and when they find the pulse above 90 they say so; but when the pulse is below 90 on a single examination I wonder how much importance should be placed on variations above and below 80.

The male risks, which are not shown on the screen, show a very good mortality with some increase in the underweight group.

In this whole group, you see again that the young risks are certainly no better than the old; there being only one exception to this in all the sub-classifications.

These are the histories of toxic goitre divided as to the presence or absence of exophthalmos. These cases all admitted having had at some time in the past goitre with hyperthyroidism. The group is very small and we must not draw any conclusions from it. Although the mortality is not nearly as high as expected, I show it because it fails to give a higher mortality in the toxic diffuse or exophthalmic goitre group. The Medical

TABLE 3

Mortality of Risks with a History of Toxic Goitre
Ratio of Actual to Expected Deaths by American Men Mortality Table*
Cases with and without Exophthalmos, Separately

	Number of cases	Years of life exposed to risk	Actual deaths	Per cent. actual of expected deaths	Average length of life since operation**		Average age at entry	
				A.M. Sel. & Ult.	All cases	Deaths	Males	Females
All cases	455	2309.79	11	91.1	10.2	8.6		
Without exophthalmos	282	1356.80	8	115.9	9.4	6.5	30.8	31.1
With exophthalmos	173	952.99	3	57.9	11.3	14.5	33.4	32.5

AVERAGE AGE AT ENTRY: ABOUT 32

*Select and Ultimate Basis
**Calculated from date of operation to death, lapse, or closing date of study

Causes of death:

Influenza or Pneumonia	2	Atrophy of liver	1
Cancer	2	Dysentery	1
Accident	3	Septicemia	1
		Angina pectoris	1

Impairment Rating Guide, you will remember, provides a higher rating for those who give a history of toxic diffuse goitre than for those with a history of toxic nodular goitre.

There is one error among the causes of death. Disease of spinal cord should read angina pectoris.

The surprising thing about this small group is that not one of them died of goitre and only one of disease of the heart. Two died of pneumonia, 2 of cancer, 3 by accident, and one each of dysentery, septicemia, atrophy of the liver and angina pectoris.

TABLE 4

Actual and Expected Mortality from Specified Causes of Death among **Females** with Non-Toxic Goitre, Present on Examination.

Cause of death	Exposed to risk	Actual deaths	Per cent. actual of expected deaths	
Cancer				Site of cancers:
Ages 35-64	10735.51	17	129	Thyroid 2; Female genitals 6; Breast 5; Stomach 3
Tuberculosis	36334.03	29	121	
Under 25	11259.68	14	146	
25-34	14300.92	12	120	
Puerperal disease	36334.03	17	133	
Ages 25-34	14300.92	12	168	
Thyroid disease	36334.03	12	1132	Following operation 4; Hyperthyroidism 7; Toxic goitre and appendicitis 1
Chronic heart disease	36334.03	9	78	

AVERAGE AGE: 26.8 AT ENTRY

This shows the principal causes of death in females with non-toxic goitres. They all had enlarged thyroids when they were insured. I showed you the mortality of this group in Table 2—87.3 A.M. Here the five principal causes of deaths are compared by number with those to be expected in our Ordinary issues, the result being expressed as the ratio of actual to expected.

Deaths from cancer, tuberculosis and especially puerperal diseases were a little above the expected, and the latter are not surprising in a young goitre group. Deaths from thyroid disease were very high, over 11 times the normal. These probably represent only a small proportion of those who became toxic after being insured as, doubtless, many were successfully operated. *The small number of heart deaths is striking* and should be given consideration by those who believe that any goitre, even the non-toxic, produces some kind of destructive cardiac poison. Perhaps, in a more aged group, the heart deaths would come up to normal, but they don't here and they did not in the Medical Impairment Study where deaths from organic diseases of the heart were less than the expected.

Dr. Lahey has shown us what excellent results can be obtained clinically in the very sick goitres, which he described. Other surgeons, too, have had good results.

Coller and Potter at Ann Arbor, reporting a follow-up of 61% of 1200 cases, find complete rehabilitation in 90% of their exophthalmic goitres and in 95% of their toxic adenomata. (See cards.)

Now all of this to me does not look as though a goitre, even a toxic goitre, produced *both* dangerous and permanent injury to the heart. It may well be dangerous, but I doubt its permanency.

Our group as a whole gives the higher mortality at the younger ages, and in the main the Medical Impairment Study agrees with this result. Now, if non-toxic goitre affects the heart, should it not show this effect more especially at the older ages, giving them the higher mortality? And wouldn't you expect this whole group to show more heart deaths? Leaving aside excessively large or substernal goitres, which can embarrass the heart and respiration merely through size and location, is it not more reasonable then to consider a non-toxic goitre as a threat which does not harm until it attacks in the shape of hyperthyroidism?

Although our group of cases with a history of toxic goitre was very small, only one heart death had been recorded up to 1931 out of 455 cases.

In this study, I see nothing that fails to agree with Dr. Lahey's conception of the relationship of goitre to disease of the heart.

DR. CHRISTIERNIN—Are there any questions that you wish to ask Dr. Lahey at this time?

DR. OLD—I would like to ask if he noticed either hypertrophy or cirrhosis of the liver in cases of hyperthyroidism.

DR. LAHEY—No. I think the only effect of hyperthyroidism that one can count on in the liver—and that is very consistent—is lessening of the glycogen reserve with an increase in toxicity. We have never seen any direct effect on the liver other than that.

I would like to say just a word in addition to what I have already told you. I think the classification of thyroids is the most confusing thing I know of in surgery. It has been mixed up, because the pathologists have had a fling at it, the surgeons have had a fling at it, and the endocrinologists have had a fling at it. (Laughter.)

Now, we all ought to talk about the thyroid in the very simplest terms and we ought to stick to it What can happen to a thyroid? If you live in a region where there is less than one part of iodine to one billion of water, you are in a region where goitre is apt to be endemic. What is the first evidence of endemic goitre? Diffuse, soft, symmetrical enlargement of the entire gland. That is the so-called diffuse colloid goitre. If it goes on long enough, then you have the nodules characteristic of the endemic goitre of Cleveland, Buffalo, the Mississippi Valley, the Northwest, the mountainous regions of Tennessee, North Carolina, and so on. That deals entirely with the goitres that arise in regions where it is endemic and which are probably related to iodine in the water. That is one group.

Now, then, there occur in thyroid glands tumors, adenomas, just like those of a female breast. This is discrete, movable, varies in size from the smallest single tumor of the size of one's little finger nail up to the size of a large water melon. These can become toxic or malignant.

Then we come to Graves's Disease or exophthalmic goitre. That is characterized by a predominance of eye signs, by a firm small gland, by occurring in youth, under forty as a rule, and by the typical picture of the four cardinal signs, many of which are often lacking, exophthalmos, tachycardia, goitre, and tremor.

That is all that there is to thyroid—diffuse, colloid goitre, the first evidence of any enlargement in endemic regions. Then nodules, which characterize the discrete adenoma. These sometimes become toxic. Then exophthalmic goitre and malignancy, of course. Malignancy never, in our experience, occurs in exophthalmic goitre, only in the tumor goitres, malignancy being carcinoma or sarcoma. This represents the whole group. We can classify them into these and they are very simple.

Dr. Bartlett—Is there any evidence that exophthalmic goitre is more common in the areas where goitre is endemic than in New England?

Dr. Lahey—If you live in Cleveland where you have many multiple adenomatous endemic goitres, one has a better chance of becoming toxic than if you live in Boston.

Dr. Anderson—Is there any group of toxic goitre in which you would advise the use of X-ray or radium in preference to operation?

Dr. Lahey—That is a contentious point. (Laughter.) I would say this, and I think this will answer the question. The most scientific piece of work which has been done on X-ray treatment of exophthalmic goitre in this country was done by Holmes and Means at the Massachusetts General Hospital. They did everything to make it fair. They appointed a committee, con-

sisting of a physician, a surgeon, a man who is interested in basal metabolism, and a roentgenologist. They looked the cases over, tabulated them, followed them well by metabolisms over a period of years and any reasonable-minded man must admit that it is possible in a definite number of cases to bring the metabolism to normal with X-ray. The great difficulty of the whole situation is, when you operate on a goitre, you remove the amount of tissue necessary to bring about a cure based upon the kind of tissue it is when you see it. That you cannot do with X-ray The disadvantage—incidentally Holmes and Means in the Massachusetts General Hospital have, I believe, largely given X-ray therapy in the treatment of exophthalmic goitre—the disadvantage is too many recurrences. It takes too long to to get them well and it takes too long to find out whether or not they are going to be well. While you are giving them X-ray to find out whether or not they are going to be well, you can have them well and back at work by surgery.

Dr. Lahey (continuing)—Are there any other questions? If not, I would like to answer one or two points and then I am through.

I would like to say definitely concerning thyroid hearts that I think the presence of thyroidism definitely indicates a damaged heart and I think there is no question, based on our experience, that these patients with undamaged hearts who are relieved of their thyroidism have just as good hearts as before. All of these patients that we followed at the end of five years have no heart lesion. They do not have myocarditis; they do not have valve damage; they do not even have conduction damage. Their hearts are just as good as before after you get rid of the hyperthyroidism. I would warn all examiners that the patients who have toxic adenomas are worse risks than patients who have exophthalmic goitre, not because it has any peculiar property of doing more damage. They are worse risks because exophthalmic goitre most commonly occurs up to forty while toxic adenomas occur from forty on. After they have lived quite a while, they develop adenomata and it then takes

time to become toxic and that means years and so the patients with toxic adenomas run much higher operative rates. The poor risk, I think, is because it occurs later in life.

Non-toxic goitre does not produce heart damage. We are very apt to confuse the German "kropfherz" that they speak of, which produces the so-called goitre heart, with ours in toxic cases. That is not so. That is the non-toxic adenoma which obstructs the trachea. Those patients who have a large goitre which is obstructing the trachea do get heart damage. That is a mechanical obstruction. One can safely say that the mere presence of a non-toxic adenoma without tracheal obstruction presupposes no question of heart damage.

DR. CHRISTIERNIN—Dr. Lahey, may I express the thanks of the Association to you for coming here and giving us that fine talk on thyroid disease and also for showing us the film. Thank you very much.

The next paper, by Dr. Blatherwick, is "Field Glucose Tolerance Tests with Determination of Blood Sugar in the Home Office Laboratory".

FIELD GLUCOSE TOLERANCE TESTS WITH DETERMINATIONS OF BLOOD SUGAR IN THE HOME OFFICE LABORATORY

By N. R. Blatherwick, Ph.D. and Mary E. Ewing, A. B.

(From the Biochemical Laboratory of the Metropolitan Life Insurance Company.)

It has become increasingly desirable for life insurance companies to have a method of collecting small samples of blood for the estimation of the sugar content in connection with glucose tolerance tests performed in the Field. The prime requisite of such a method is the preservation of all the sugar. Furthermore, the average medical examiner must be able to collect the samples of blood.

This problem has been investigated by us for some time but without adequate success until the summer of 1931. A satisfactory preservative for the blood sugar was found and a method of collecting the blood was evolved which has proved its usefulness. The examiner collects more blood than is required for the determination and the exact measurement is left to the laboratory analyst. The blood is collected in the pipettes which are used for the final measurement.

A paper by Miss Ewing describing the method of collecting and preserving the blood samples will be published in the near future in the Journal of Laboratory and Clinical Medicine. For the sake of completeness, the essential parts are given below.

A 0.1 cc. capillary pipette lined with preservative salts is used for the collection, the blood being sealed inside the pipette by means of a rubber band stretched over the ends. For this purpose the usual Folin micro-blood pipette (1) is shortened by cutting off the blunt end, and an extra graduation is added about 3 cm. beyond the 0.1 cc. mark. The blood sample is collected to this second graduation and the measurement is left to the analyst at the time of testing. The procedure is as follows: Make the lancet puncture in

the finger-tip, allow the blood to flow into the pipette, tip it back and forth a few times to dissolve the preservative lining and attach the rubber band. At the time of making the analysis measure the sample by discarding the excess blood and analyze it in the same way as a fresh sample.

The pipettes selected were 16 cm. long and it was found that the corresponding rubber bands must be 1 cm. in width and 9.5 cm. in length. If longer or narrower the bands did not fit the pipettes snugly and permitted some evaporation of the blood column inside. If shorter they were too tight and might be punctured by the tips of the pipettes.

As a preservative Lax and Szirmai (4) recommended the use of sodium fluoride and mercuric chloride. According to their report these two salts, in the proportion of sodium fluoride, 1.0 per cent., and mercuric chloride, 0.1 per cent., prevented glycolysis in blood samples for periods up to a month and had no reducing effect on the testing reagents.

Preparation of Suspension-Solution

Dissolve 0.5 gm. of sodium fluoride in 15 cc. of water and rinse into a 50 cc. volumetric flask, using as little water as possible for the rinsing. Add 95 per cent. alcohol until the flask is about half full and the sodium fluoride is brought down as a fine precipitate.

Dissolve 0.5 gm. of mercuric chloride in 3 cc. of 95 per cent. alcohol and add this to the fluoride suspension, making up the volume with additional alcohol.

The preservative thus prepared will show only a slight deposit of mercury and will be suitable for use for at least a month if kept in a dark cupboard.

Preparation of the Pipettes

Shake the suspension thoroughly, then pour two or three cc. into a small beaker. Agitate the beaker to make sure that the salt is uniformly dispersed and draw the liquid and suspension into the pipette to the second graduation. As many pipettes as desired may be filled at one time, the suspension in the beaker being stirred well

with each pipette before it is filled and the pipettes laid horizontally after filling. Each pipette in turn is then tipped slightly toward the blunt end so that the suspension particles are drawn away from the tip, then tilted slowly toward the tip so that the liquid begins to flow away from the salt. When the liquid reaches the opening at the tip of the pipette apply a cloth or filter paper to draw it off, being careful not to absorb any of the salt. If the precipitate runs toward the tip too swiftly tilt once more toward the other end before continuing to decant at the tip. It is easier to see the precipitate and to regulate its progress if the pipette is held in front of some black surface as a background. When the salt is concentrated into a space a little less than a cm. in length at the tip of the pipette direct a gentle current of air through the tip from a rubber tube. This will cause the wet salt to flow back along the bore of the pipette. Care must be taken to see that none is blown out the opposite end. Continue the gentle blowing until the salt is dried in a thin, even lining for the pipette. It requires only about a minute's time to line a pipette in this way and once prepared and dried it may be kept indefinitely before using.

Collection of the Blood

Make the lancet puncture in the finger-tip and collect the blood in the usual way for the micro method, allowing it to flow into the pipette to the second graduation and being careful not to admit bubbles. Then tip the blood column from one end to the other in the pipette, three times in each direction, in order to dissolve the preservative. Finally let the blood run down to the tip again, insert the tip of the pipette into a fold of the rubber band and stretch the band over the opposite end, pressing it down snugly over the blunt end. If the edges of the blunt end are not well covered some evaporation of the blood column toward that end may occur.

The bands should be washed with water and wiped dry before using.

If the first drop of blood fails to enter the pipette readily, wipe the tip with a cloth moistened in alcohol.

Making the Determination on Preserved Blood

Remove the rubber band and with a dampened cloth wipe off any blood on the outside of the pipette. Then with a slender wire such as the one inside a syringe needle, loosen the blood inside the tip. With the tip end held up, shake the pipette downward until the blood column begins to fall, then reverse the pipette and allow the blood to flow toward the tip again. Repeat this process until the blood cells, which may have settled along one side or at the end, are again mixed with the plasma. Then run the blood down to the tip once more, lower the meniscus to the 0.1 cc. mark and blow the measured blood into the diluting fluid. Rinse the pipette repeatedly. Finish the sugar determination according to the Folin Method (1,2,3.).

The ease with which the blood begins to flow depends to some extent on the length of time since it was collected and also on the weather. The blood will remain more loose and fluid in the pipette at lower temperatures than during the very warm months.

In some cases it will be found that small patches of blood will adhere to the inside of the pipette and the rinsing is then very important. It will be found either to dislodge the blood or to extract its glucose. A stiff piece of rubber tubing, four or five cm. in length, may be conveniently attached to the pipette to serve as a mouth piece for the rinsing.

Experimental Results and Discussion

A series of blood specimens were kept from one to fourteen days and showed sometimes a slight drop, as often a slight increase over the corresponding fresh samples. The practice was to collect two samples of the same blood, one to be analyzed at once, the other to be kept in the pipette. Out of 88 comparisons made during the spring and summer of 1931, only 11 showed an error of more than 5 per cent. The greatest error encountered was 7.5 per cent. The figures for these comparisons are given in Table I.

The keeping period is apparently limited only by the beginning of evaporation of the blood column. It is easy to tell by inspection

if evaporation has occurred to any extent. If so, the value will tend to be high. It has seldom been noted under eight days even in warm weather and showed only very slightly after fourteen days in the winter.

The procedure was adopted by this Company for tolerance tests in October, 1931, and has been used since then with acceptable results in all cases where the collection was made according to instructions. A grooved wooden box holding the pipettes, bands, and a lancet is placed inside a pasteboard carton for mailing[1]. Six of these boxes, containing three samples each, were sent to San Francisco to be returned as a test of their travelling qualities. Four of the boxes showed the samples in as perfect condition on return as when mailed. In two of the boxes a slight separation in the blood column toward the blunt end of the pipette occurred. This did not interfere with the analysis, since the separated blood could be tipped out before the sample was measured. Results of the analyses of these samples are given in Table II.

[1]The outfits now in use were made especially for the purpose by the Empire Laboratory Supply Company, New York, and satisfactory rubber bands were obtained from the Hodgman Rubber Company, Malden, Massachusetts.

Procedure used by the Metropolitan Life Insurance Company in performing Field Glucose Tolerance Tests.

The medical examiner is sent a grooved, wooden box containing four pipettes and a lancet together with a data sheet, directions for performing the test, and a gummed label to be affixed to the mailing carton when it is returned to the Home Office. This package is sent by first-class mail and requires 36 cents postage. Copies of the data sheet and of the directions follow:

DATA SHEET

METROPOLITAN LIFE INSURANCE COMPANY

BIOCHEMICAL LABORATORY

GLUCOSE TOLERANCE TEST
with Estimations of Blood Sugar

Applicant's name *(print)*

Name of District

Date 193

Put X mark in box below opposite kind of case for which specimens are secured	
ORD. OR INT.—New App.	
ORD. OR INT.—Postponed	
ORD. OR INT.—Revival POLICY NO.	
CHANGE APPLICATION POLICY NO.	
ACCIDENT and HEALTH	
INDUSTRIAL	
F.1145 NEW EMPLOYEE	
M.D.52 YEARLY EXAM.	

Sugar Tolerance (50 grams glucose)

Three specimens of blood are to be obtained, the first just before the administration of glucose, the second thirty minutes and the third two hours after the glucose is given. A specimen of urine is to be obtained before the administration of glucose and another two hours after the glucose is given. Record the actual times that these various specimens are obtained.

Start the test in the fasting condition or at least two and one-half hours after eating. State which

First specimen of blood, before glucose time

50 grams of glucose given time

Second specimen of blood, 30 minutes after glucose time

Third specimen of blood, 2 hours after glucose time

REMARKS:

First specimen of urine, before glucose time

volume in cc.

Second specimen of urine, 2 hours after glucose time

volume in cc.

Send samples of the specimens of urine to the Home Office in the usual containers.

Medical Examiner

When this sheet has been filled out, send it, together with the pipettes and lancet, to the Home Office. Affix the addressed label to the mailing carton in which you received the case.

Directions for Drawing Blood for Glucose Tolerance Test

BIOCHEMICAL LABORATORY
METROPOLITAN LIFE INSURANCE COMPANY

READ DIRECTIONS CAREFULLY BEFORE STARTING TEST

You are furnished specially prepared pipettes, containing a preservative, for the collection of specimens of blood. They are identified with the different specimens of blood by etched bands at the ends of the pipettes. Use the pipette with one etched band for the first specimen, that with two bands for the second and that with three bands for the third specimen of blood. The fourth pipette, having no etched bands, may be used in case of emergency.

Before beginning the test, examine the lancet. Draw back the plunger and release it to become familiar with its operation. *Do not readjust the blade.* This is set at the right length for a good puncture.

Dip the blade end of the lancet in alcohol and set the plunger ready for the puncture.

Place the bottom part of the case containing the pipettes on an ordinary sized book or on the inverted top part of the case. Rest the pipette in the middle groove of the case with the tip end supported by and extending about an inch and one-half beyond the groove in the end of the case. *The blood will flow in rapidly and without bubbles with the pipette at this angle only.*

Seat the applicant with his left forearm and hand, palm upward, resting on a table.

Wipe the tip of the finger, preferably the fourth, with alcohol.

Rub the tip vigorously where the cut is to be made, so as to insure an ample supply of blood, and wipe dry.

Hold the tip of the finger tightly between the thumb and forefinger.

Press the tip of the lancet firmly into the flesh of the finger tip and release the plunger. See Figure I.

Wipe the cut dry.

Grasp the applicant's finger with the left hand and, with the thumb and forefinger, press out a drop of blood.

Move the case until the small opening of the pipette just touches the surface of the blood. See Figure II.

Do not touch the finger with the tip of the pipette.

Do not change the angle of the pipette. The drop of blood will flow in from capillary attraction.

FIGURE I. *Application of Lancet*

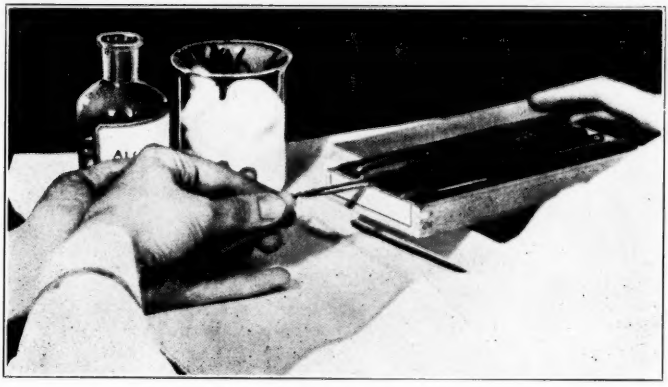

FIGURE II. *Filling the Pipette With Blood*

If the blood should not start to flow into the pipette, rotate the pipette until the flow begins. *Do not use suction.* If the first drop of blood fails to flow in readily, wipe the tip of the pipette over the opening with a tuft of cotton moistened in alcohol.

Press out a second drop of blood and proceed as before.

If the blood flow is inadequate, milk the finger back of the first joint toward the tip.

Rub the cut with cotton or gauze between applications of the pipette, if necessary.

Continue collecting the blood, *one drop at a time,* until the *pipette is filled to the second graduation mark, which is colored red.*

Immediately tip the blood from one end of the pipette to the other, THREE TIMES IN EACH DIRECTION, FINISHING WITH THE BLOOD EXTENDING TO THE TIP OF THE PIPETTE.

Insert the tip of the pipette into a fold of the rubber band and stretch the band over the blunt end. See extra pipette with rubber band in place.

It should not take longer than thirty seconds to fill a pipette.

It is very important not to admit bubbles into the pipettes. If a bubble forms in a drop of blood on the finger tip, wipe the drop away and press out another. If a bubble gets into the pipette, tip it out before finishing the collection. The blood must be in *one unbroken column.*

FURTHER PRECAUTIONS:

Do not contaminate the pipettes with glucose.

Do not tamper with the pipettes in any way. They contain a measured amount of preservative.

Be especially careful not to touch the tips of the pipettes before they are filled.

Use alcohol, only, to clean the finger.

Caution the applicant to eat nothing while the test is in progress.

EXPERIENCE WITH THE METHOD

We began using this method October 1, 1931, but it was not placed on a sound operating basis until December 1, 1931. In the eight months' period since the first of last December, 540 requests for this test were made. 201 (37.2%) of the applicants refused to take the test. 41 (13.8% of those who tried) examiners failed to make satisfactory collections of the blood. 255 (47.2% of those requested) satisfactory collections were made. Of the latter, the timing of the specimens was correct in 211 (82.7%) cases, and it

TABLE I.

A Comparison of the Glucose Values of Fresh Samples and Preserved Samples of the Same Bloods

No.	Days kept	Glucose in milligrams per cent.		Per cent. of deviation	No.	Days kept	Glucose in milligrams per cent.		Per cent. of deviation
		Fresh Sample	Preserved Sample				Fresh Sample	Preserved Sample	
1	5	217.5*	213.9*	1.7	45	10	136.9	141.8	3.6
2	5	100.8*	97.1*	3.7	46	8	96.6	94.8	1.9
3	7	84.9*	86.8*	2.2	47	8	202.0	212.7	5.2
4	1	90.1*	90.9*	0.8	48	8	105.8	100.0	5.5
5	6	189.5*	192.4*	1.5	49	10	100.0	105.2	5.2
6	6	229.9	227.3*	1.1	50	7	137.9	130.7	5.2
7	6	94.6*	99.0	4.7	51	6	101.0	102.0	1.0
8	6	152.6	150.4	1.4	52	6	210.5	219.7	4.4
9	6	132.5*	137.9	4.1	53	5	163.9	166.6	1.6
10	6	98.0	101.5	3.5	54	11	94.3	90.9	3.6
11	6	206.1	202.0*	2.0	55	10	138.8	132.4	4.6
12	6	135.1*	132.9*	1.6	56	10	169.4	167.3	1.2
13	6	91.9*	93.9*	2.2	57	9	99.5	101.5	2.0
14	4	176.9	182.9	3.4	58	9	200.0	206.1	3.1
15	4	92.0*	95.1*	3.4	59	9	175.4	166.6	5.0
16	3	183.4	183.4*	0.0	60	9	162.5	162.5	0.0
17	6	168.0	169.4	0.8	61	9	104.7	105.2	0.5
18	6	96.6	97.6	1.0	62	6	204.0	215.0	4.9
19	6	142.7	150.3	5.3	63	10	119.7	121.8	1.8
20	6	90.1	83.3	7.5	64	10	113.6	115.6	1.8
21	6	120.4	124.2	3.2	65	6	112.3	110.5	1.6
22	6	178.5	175.4	1.7	66	6	90.5	90.1	0.4
23	6	200.0	190.4	4.8	67	6	156.2	149.2	4.5
24	6	133.3	129.0	3.2	68	6	190.4	183.4	3.7
25	4	241.0	232.5	3.5	69	8	103.1	101.0	2.0
26	6	181.8	176.9	2.7	70	12	104.1	105.8	1.6
27	6	145.9	141.8	2.8	71	11	192.4	198.0	2.9
28	12	95.7	99.0	3.5	72	7	139.8	132.4	5.3
29	6	90.9	86.2	5.2	73	5	91.3	96.1	5.3
30	6	145.9	151.4	3.8	74	6	155.0	153.7	0.8
31	6	130.7	131.5	0.6	75	6	91.3	89.7	1.8
32	6	114.9	116.9	1.7	76	6	91.3	91.3	0.0
33	1 hr.	71.6	69.7	2.7	77	6	145.9	152.6	4.6
34	6	95.2	97.6	2.5	78	5	99.5	94.8	4.7
35	10	124.9	131.5	5.3	79	10	181.8	186.9	2.8
36	8	105.2	105.2	0.0	80	6	175.4	168.0	4.2
37	8	212.7	212.7	0.0	81	6	266.7	274.0	2.7
38	8	247.0	250.0	2.0	82	6	303.0	317.5	4.7
39	8	175.4	170.9	2.6	83	6	259.7	253.1	2.5
40	10	119.0	114.9	3.4	84	10	112.3	109.8	2.2
41	9	149.2	147.0	1.5	85	7	166.6	168.0	0.8
42	11	93.0	87.3	6.1	86	9	144.9	139.8	3.5
43	8	100.5	104.1	3.6	87	7	103.6	100.5	3.0
44	8	178.5	186.9	4.7	88	4	162.5	162.5	0.0

*This figure is an average of duplicate determinations.

was incorrect in 45 (17.3%) cases. A normal blood sugar curve was found in 162 (63.5%) and an abnormal curve in 93 (36.5%) instances. In 55 (21.6%) cases, a normal blood sugar curve was found with at least one of the two specimens of urine showing more than 0.4 minus per cent. sugar. 32 (12.6%) cases having an abnormal blood sugar curve had 0.3 per cent. or less sugar in the urine. There were four instances in which one or more pipettes was broken in the mail and another outfit was requested. In order that there may be no misunderstanding, it may be stated that the

TABLE II.

The Glucose Values of Blood Samples Sent Through the Mail and of the Corresponding Fresh Samples Collected at the Same Time

Box No.	Date Sent.	Date Analyzed.	Glucose Milligrams per cent.	
			Mailed Sample.	Fresh Sample.
1	Feb. 11	Feb. 19	92.6	97.6
			183.4	180.1
			66.8	68.5
2	Feb. 15	Feb. 29	129.0	129.8
			124.9	
			129.0	
3	Feb. 17	Feb. 29	90.9	94.8
			212.7	215.0
			92.6	100.0
4	Feb. 17	Feb. 29	155.0	157.4
			156.2	
			162.5	
5	Feb. 17	Feb. 29	88.9	88.9
			176.9	180.1
			50.3	56.3
6	Feb. 24	Mar. 4	91.7	90.9
			169.4	161.2
			107.0	110.5

reason the above figures do not total up to 100 per cent. is that final disposition has not been made of a certain number of the requests.

Remarks Concerning the Method

Our experience shows that about 14% of the examiners who have tried to collect the blood specimens have failed. This failure is certainly caused by not following the directions for drawing the blood. The most likely source of error is in allowing the tip of the pipette to touch the finger and not the surface of the drop of blood. Another rather common mistake is failure to mix the blood with the preservative. Such bloods can be used providing the values yielded show hyperglycemia. The presence of air bubbles in the pipette does not necessarily mean the loss of the specimen. Such samples, after they have been mixed in the usual manner, can usually be transferred to a fresh pipette by allowing the blood to flow from the tip of the filled pipette into the tip of the empty pipette. Whenever an air space is encountered, the original pipette is removed and the blood is allowed to flow until it again reaches the tip, when it is applied to the other pipette as before. The incorrect timing of the various blood specimens is a matter of no small importance especially that of the third specimen two hours after the administration of the glucose. Some of the deviations from the correct times may occasionally be unavoidable, but every effort should be made to obtain the samples at the scheduled times.

The appointment of one examiner in each center to perform all glucose tolerance tests would, no doubt, practically eliminate the rather small percentage of failures which we have encountered. The designation of some laboratory to perform the test would be a still better arrangement. It is understood that the blood specimens would always be analyzed in the Companies' laboratories.

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Dr. Blatherwick—I am tempted to cover the essential points of this contribution in the printed paper and therefore will not take much of your time this morning. I am passing around samples of the outfits which we use at the Metropolitan in the collection of samples of blood from the field. This outfit, you will see, consists of the box, containing four pipettes and one small lancet. We wish to collect three specimens of blood—one before the glucose is given, one thirty minutes after the administration of glucose and, third, two hours after the glucose has been given. We have sent an extra pipette in case of emergency of any kind and the extra pipette also has the rubber band in place, showing the proper way to seal the pipette after the blood has been collected.

These pipettes are marked with bands at the blunt end. One pipette has one band. That is to be used for the first specimen of blood. The second has two bands. That is for the thirty-minute specimen. The third has three bands, which is for the two-hour specimen. You will note that the pipettes contain a deposit of salts. These are for the purpose of preventing coagulation and the disappearance of the blood sugar. The pipettes have two graduation marks; one is the graduation mark used in the determination by the laboratory and the second graduation mark is colored red and the blood is to be collected to the second graduation mark.

The manner of making the collection is to take the pipette and place it in the center of the box with the tip end of the pipette extending through the groove at a convenient distance of an inch and a half or so and the whole box may be raised from the surface of the table. Then the finger is punctured with the lancet after sterilization with alcohol. The tip of the pipette is placed to the drop of blood, when the blood will flow automatically into the pipette. After the pipette is filled to the second red graduation mark, then it is tipped toward the blunt end of the pipette and then back three times in each direction in order to mix the blood with the preservative. It is then sealed with the rubber band. Bloods collected in this way keep the sugar in a very nice manner. You will notice from the

figures in the tables that the bloods have been kept up to ten days easily with the original amount of blood sugar present.

Now, our actual experience with this method, which has been used now for just about a year, shows that the average medical examiner is able to make this collection satisfactorily from all points of view in better than 80%, 86%, that is, 14% of the examiners who have tried to make the collection have failed to do so for one reason or another. The most obvious reason is that the tip of the pipette is allowed to touch the skin or the finger and not the drop of blood. Of course, if you do that, it stops off the chance of the blood to flow into the pipette, because you block the opening of the pipette. Another difficulty encountered is that some of the examiners failed to mix the blood after it has been collected properly.

Usually those bloods are fluid and can be handled all right, but we don't wish to make any mistake and we do not report the sugars found in those bloods unless they happen to be hyperglycemia. We are not sure whether the normal amount of blood will keep under those conditions, but if the blood sugar is high, then we of course save them. Occasionally we find an extra ambitious examiner who completely fills the pipette and therefore the sample is not satisfactory because he is adding more blood than we counted on for the amount of preservative.

Personally, I have been quite well satisfied with the way this has turned out, this absolutely new technique we are giving our examiners, and I thought we would have a good many more failures than 14%, so that I am really quite satisfied.

I might say that this outfit has a cover which I didn't pass around, because I wanted to make sure the pipettes wouldn't fall out. This outfit has a cover which fits right on over the top. Then we merely place a rubber band around here, put it in the mailing carton, in which the outfit was originally mailed. This contains the data sheet and the directions for making the test and the return label to be fixed to the carton for the return journey. The whole thing goes in this carton and is mailed at first class rates, which, under present conditions, costs 36 cents. I believe that is all I have to say.

DR. CHRISTIERNIN—Dr. Folin, will you please discuss Dr. Blatherwick's paper?

DR. FOLIN—I have not written any comments on this paper. It is so simple and so clear to me, at least, that is does not seem to call for any except that I would say that it is evidently a very clear and careful and probably very important piece of work tending to meet a very serious problem that has, of course, been with us now for a number of years, namely, the problem of how the average physician is going to get the benefit for his patients of the undoubtedly useful analytical technique which is now available in the field of blood analysis. This innovation of Dr. Blatherwick's seems to me a good one and at least one line of attack on this important problem.

I might say in this connection that I ran into another field of blood analysis during this Summer during a little trip which I took into Sweden. This has to do with the determination of alcohol in blood. The whole country there is very much devoted to the use of alcohol in various forms, and there is no objection to it. (Laughter.) The people get all the beer and all the wine they want, and in addition to that, up to 4½ quarts of 96% alcohol per month, which seems to be adequate for most of them. (Laughter.) Also there is no objection to the amount that they take within the prescribed limits, nor is there any objection to their driving an automobile or a motorcycle while under the influence of all that liquor. But yet, if a person who has imbibed too much runs into an accident, the question comes in whether he was intoxicated or not, and that is the difference between them and us. That fact not only greatly increases the presumption of his guilt, but it also brings with it a very much more severe punishment than if he had been sober. Now the point I come to is as to how they determine whether the man was sober or not. They do that by means of blood analysis. The various people involved in such an accident at once are taken to the police station, or perhaps to the hospital, and the first thing which is done is that a sample of finger blood is taken from him. That small sample is sent to the central office in

Stockholm. There the alcohol is determined and on the basis of the findings, the man is either sober or drunk. The test is so sensitive that if one takes one glass of 3% beer, it will show that there was alcohol in the blood. Therefore, the determination has to be quantitative and a definite limit has been set which shows some intoxication, and, of course, the test being quantitative, it also shows varying degrees of that state.

The point I am coming to is that if they can consider it worth while to have a mail order business in quantitative blood analysis for the sake of proving whether a man is drunk or sober, I think we ought to be able to use it for the more serious purposes of life insurance, and I hope also for the more widespread need of the medical profession in general. I think that this line of attack of sending blood through the mail certainly represents one important attack on that problem.

I am fairly well in touch with the situation and have seen for a number of years the keenness with which doctors receive every simplified method for the making of such determinations, and it is a question of how they are going to get the benefit of the results. Many of them are, of course, trying to do the work themselves, but one is a little bit afraid of their results, with their meager office facilities, their lack of chemical experience and, above all, their lack of time, and one is a little afraid, of course, of just how the results might be some time. In quantitative work, it must not be forgotten that poor results, very poor results, are worse than none at all.

Just a few weeks ago here this Summer, a new experiment was being made in Massachusetts in that a new colorimeter, which is very inexpensive, has been turned out and with it a set of all the needed standard solutions. Two hundred and fifty thousand were made at once before any attempt was made to sell them and all of them were sold within a short time, so that the supply is now exhausted, showing how widespread is the hope and the need of the medical profession for getting hold of these simple devices to help them in their study of cases.

There is one aspect in Dr. Blatherwick's paper that perhaps it may be worth while to call to your especial attention, and that

is the times when the blood samples are taken—one sample before the giving of the sugar, another thirty minutes later and the final sample two hours after the taking of the sugar. These seem to me to represent the very best than can be done on the basis of 50 grams of glucose. I mention these facts because I had occasion a short time ago to see the results of a questionnaire sent out by Dr. ————, from which I learned that the practices were very far from uniform among the different life insurance companies. It seems to me that it would be better if you settled down to some more uniform definite conditions, and I think that these conditions mentioned by Dr. Blatherwick are as good as any that you can make, and I say this although personally I don't use glucose any more. I still prefer very much to use cane sugar. We have again tested our cane sugar and find that 75 grams are quite equivalent to 50 grams of glucose, and will give results similar to it in every way. I have done this on normal persons and Dr. Joslyn has, at my request, done it on patients, and his conclusions coincide with mine that 75 grams of cane sugar will give substantially the same results as 50 grams of glucose. That is, however, just a small minor point.

I think that the conditions laid down by Dr. Blatherwick for the making of sugar tolerance tests are so good that you are not likely to improve upon them, and I suggest that you adopt them.

There is one other minor point that occurred to me in connection with Dr. Blatherwick's work. As you notice, there is some miscarriage in the results. That is undoubtedly unavoidable and presumably will decrease as the physicians get more experience in collecting blood by means of these micro-pipettes. I wonder, however, whether it might not be advantageous to have two tubes for the two-hour period of collection. It is that two-hour period that tells the story. Personally, I care very little either about the results of the preliminary blood sample or of the 30-minute sample, but I know that you do not all agree with me there. Perhaps a few of you do, but I do know that Dr. Joslyn, for example, lays a great deal of store on the

height, the maximum height, to which the blood sugar will rise. Nevertheless, whether you agree with me or not, the fact is that the crucial figure in the experiment, such as those described by Dr. Blatherwick, is the sugar level which you get at the end of the two-hour period. Therefore, if you have two of those, even if you should take one at the expense of the preliminary sample or at the expense of the thirty-minute sample and instead of that have two for the two-hour period, it probably might reduce the number of unsatisfactory attempts. I thank you.

DR. CHRISTIERNIN—I had hoped that Dr. Benedict might have something to say on this subject also, but he tells me that he has discussed it with Dr. Folin and has nothing to add to what the latter has said. Are there any questions or discussion of Dr. Blatherwick's paper?

DR. CORT—Gentlemen: I have been very much interested in Dr. Blatherwick's paper on "Field Glucose Tolerance Tests", particularly the method he recommends for the collection of specimens. My interest is based upon personal experience over the past four years in our blood sugar work.

We have utilized a small, rubber corked bottle in collecting our specimens. This bottle holds easily .6 cc. of blood when corked and in the event of clotting our specimen is ample to meet the requirements of the test, which, as you know, utilizes .1 cc. of specimen serum.

It would appear to me that there was not sufficient sample obtained from the field to insure an accurate laboratory determination. I estimate that if the pipette is filled to the mark as suggested by Dr. Blatherwick, he gets .15 cc. of specimen—not a great excess of serum over the required amount for the test and not sufficient to make two tests if two results would be desired.

I also suggest that a bottle collecting larger specimens is preferable in the event of clotting. Clotting depends largely on the thoroughness which the fluoride preservative has been

mixed with the specimen. It would also be influenced by temperature and age. Dealing with such small specimens, the difference between .12 cc. and .6 cc. is small—at most it is represented only by a few drops of blood. Clotting in Micro chemistry is always to be anticipated even in the presence of the anticlotting preservative. When it occurs within a pipette of narrow lumen it adheres to the side walls, making a difficulty in decanting the serum and also presenting the necessity of a most thorough rinsing of the container. I assume that the separation of the clot in the specimen pipette may at times present difficulty to the laboratory technicians, at which time, of course, they then have to proceed in the same manner as we do with the bottle by decanting out the serum for the test. When this necessity arises it would appear that the bottle as a container would be preferable to a narrow pipette.

Again, under certain circumstances when the clot adheres to the side walls and when dealing in Micro chemistry, I should always feel a little bit uncertain that my washing had been complete and I had obtained the full specimen. With the larger container used by us the excessive specimens always afford sufficient material for two or more tests and the urgency of a careful washing of all clots of the specimen free from the bottle does not present itself.

The bottle, I am confident, will work out better than the pipette as a collection receptacle. It has for us and our figures of spoiled, insufficient, or unsatisfactory specimens over a period of four years show a much better figure than Dr. Blatherwick has obtained to date.

I would add that our percentage of unsatisfactory specimens has been very materially improved since we notified our Examiners that we would not pay for the collection of insufficient or unsatisfactory specimens. Examiners then took more pains in filling the bottle and thoroughly mixing the fluoride preservative in the specimen.

Dr. Blatherwick's data sheet and circular of instructions are as complete and as comprehensive as I have seen.

Dr. Rowley—May I ask Dr. Folin how much of a measure of protection, if any, we have in getting that specimen a half-hour after the glucose and in noting the fact that there is a rise in the blood sugar. If we were to omit that second specimen and not know whether there was a rise in blood sugar after the administration of sugar, cane sugar or glucose, would we be lacking some positive and valuable information to protect us against cases who might prepare themselves for a sugar load?

Dr. Muhlberg—May I ask Dr. Folin a question? We have been doing the triple blood sugar test, but I haven't felt that I have derived very much information from the first and second examinations except in so far as it assures us that the glucose has not been vomited and has been absorbed into the blood. We are thinking somewhat along the line of one blood sugar estimation two hours after the glucose and then putting a little something in the glucose itself that can be detected in the urine that is voided two hours after, feeling that we can rely on that to feel certain that the glucose has been absorbed and that the solution has not been vomited. Do you consider that a safe procedure, Dr. Folin?

Dr. Christiernin—Dr. Blatherwick, will you reply to the first question?

Dr. Blatherwick—We have experienced difficulty at times of partial clot or sort of drying of blood on the wall of the pipette, but so far we have been able to determine that by a good thorough rinsing of the pipette, we will not lose sugar that way.

Regarding the sample of blood necessary to get a reliable estimation of blood sugar, I don't think there is any question about that. The amount of blood used is .1 cc. and the amount of blood collected is .12 to .13 (something like that). Of course, when we get the sample back at the laboratory, we remove the band and put a fine wire into the tip end of the pipette to loosen it, tip it back and forth to mix it thoroughly and then just draw off the blood.

DR. FOLIN—That matter of the first two sugar collections represents necessarily a difference of opinion. As to whether the sugar rises or not during the first half hour, I think that depends only on the different abilities of perfectly normal people to take care of the absorbed sugar. We have seen two cases at different times when the student failed to get the material rise of sugar at the end of 30 minutes with 75 grams of cane sugar and we repeated it then with those same people with 50 grams of glucose and found exactly the same results. They were simply people who disposed of the sugar quickly and did not get any rise. Similarly I have noticed from Dr. Joslyn's work on his patients that same thing. He applied this cane sugar only to doubtful cases where he had made a number of preliminary tests with his regular test meal and I noticed from three of those that there was no rise of the blood sugar at the end of thirty minutes, but nevertheless those cases showed a little sugar in the urine, a sufficient rise in the urine as to indicate that he was indeed dealing with cases of renal glycosuria. Therefore, incidentally it proves that although there had been no rise, there had nevertheless been absorbed enough sugar, and personally I know of no way in which a person could manipulate it so as to fail to absorb the given sugar.

Therefore, on these grounds, I consider both the first and the second collection of blood as less important but still very interesting and would recommend that if you have the time to do it, and, of course, collecting samples of blood by mail you would never think of sending only one sample and two 2-hour samples might be better even if you had to sacrifice one of the other two.

I don't know, Dr. Muhlberg, whether coloring the sugar or doing anything of that sort will help us very much, and you can see from my remarks that I do not think that is needed. I do not think that there is any question about the fact that the two-hour result is the particular one to be depended upon. I say this without in any way implying that either one of the other two specimens should not be taken and analyzed if the

conditions permit it. I only emphasize that the two-hour sample is the one that we must depend upon.

DR. CHRISTIERNIN—This brings us to the final and very important paper by Dr. Rein on the "Kline Test" for syphilis.

One of the most difficult problems which the medical reviewer of insurance applications has to deal with is that of the applicant who admits a history of syphilis or is suspected of having had the disease. Statements of the attending physicians certifying to adequate treatment or negative Wasserman tests may be absolutely correct, but there is always a feeling of uncertainty and reluctance to accept these statements unless they come from known physicians, hospitals or laboratories of high standing. In his paper entitled "Kline Test" Dr. Rein describes in detail a method which gives the information desired accurately, inexpensively and with no inconvenience to the applicant.

DR. REIN—Mr. Chairman and Members of the Association: (Read paper.) (Also presented slides.)

THE VALUE OF THE KLINE PRECIPITATION TESTS FOR SYPHILIS

By Charles Robert Rein, M. D.
New York.

During the past several years there has been an increasingly wide spread use of various precipitation tests for the diagnosis of syphilis. This is due to the fact that the precipitation tests are simple in technic and at the same time are as accurate as the complement fixation tests. Of the various precipitation tests, the microscopic slide test developed by Kline has been found by many to be very accurate and extremely simple in technic. It moreover enjoys the advantage that it can be done with heated sera(1), unheated sera(2), defibrinated finger bloods(3) and spinal fluids (4).

The test as performed with heated sera has been clinically evaluated and serologically compared with other precipitation tests and also with the complement fixation test for syphilis by a number of observers. Littman(5) reviewed one hundred and forty-seven thousand two hundred and fifty (147,250) Kline tests, performed in some sixteen clinics in various parts of the country and found them in agreement with clinical findings in 96.52 per cent to 99.18 per cent according to the sources of material studied. He found the Kline tests to be more sensitive than the Kahn, Hinton and Wasserman tests, in various stages and types of syphilis. The false positives in general have been few in the hands of the expert serologists.

Within the past year we have had the opportunity of personally conducting several comparative studies of the Kline Tests with the Wasserman, Kahn and Hinton tests in New York City.

In one of the studies made in the Department of Dermatology and Syphilology of the Post-Graduate Hospital(6), finger bloods were employed, in a series of five hundred patients. In this

group were individuals under treatment for various types of syphilis and miscellaneous dermatoses. This test was compared with the routine Kline, Wasserman and Kahn tests performed on blood obtained from the arm. It was found that the Kline finger blood tests and arm blood tests agreed in practically 100 per cent. It was also shown by this comparative study, that the Kline tests were considerably more sensitive than the Wasserman and Kahn tests and agreed more closely with the clinical data. In addition it should be noted that there were no false positives with the Kline tests.

A further study was carried out with patients from the Venereal Disease Clinic, New York City Department of Health[7]. In this study 110 consecutive patients with sores suggestive of syphilis were employed. Seventy-four of these patients were ultimately proved to be infected with syphilis. Here again the Kline test was found to be more sensitive and became positive earlier than the Wasserman, Kahn and Hinton tests. In addition there were no false positives. These studies indicate that the Kline test for syphilis is certainly no less specific and considerably more sensitive than the Wasserman, Kahn and Hinton tests.

When the Metropolitan Life Insurance Company adopted the use of Folin's micro-method for sugar determinations they also had in mind the development of a test for the detection of syphilis with small amounts of blood. It is clear that to be ideal the test should be one that could be done with small amounts of blood easily collected and transported. Moreover, the test would have to give satisfactory results with blood even ten days old, to allow sufficient time for the specimens to be sent to the central laboratory for examination.

For reasons of technic the finger blood test referred to above is not suitable for insurance work. In order to overcome the objections, the following procedure was adopted which seems to meet all the requirements of insurance work. The detailed technic is as follows:

FIGURE 1

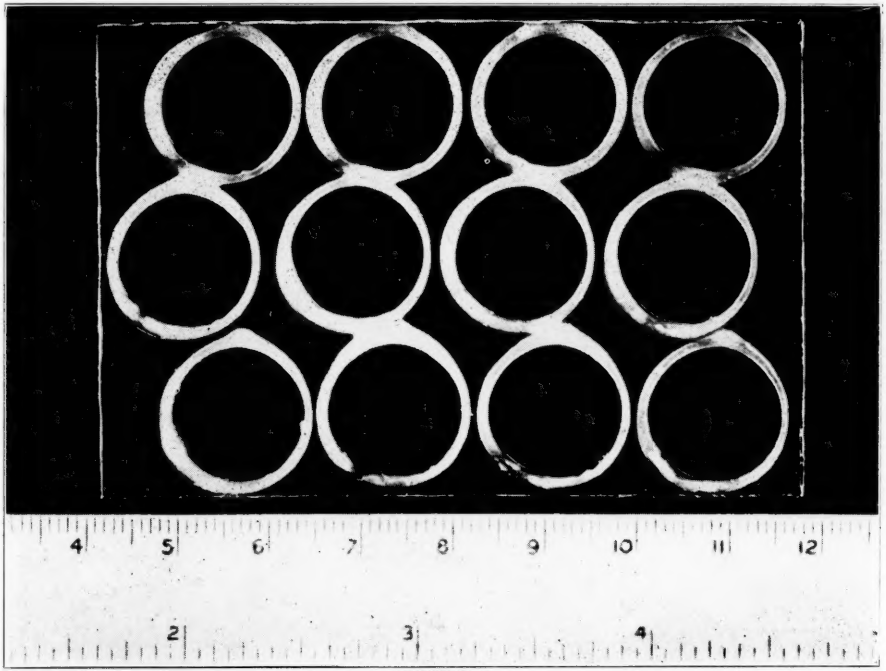
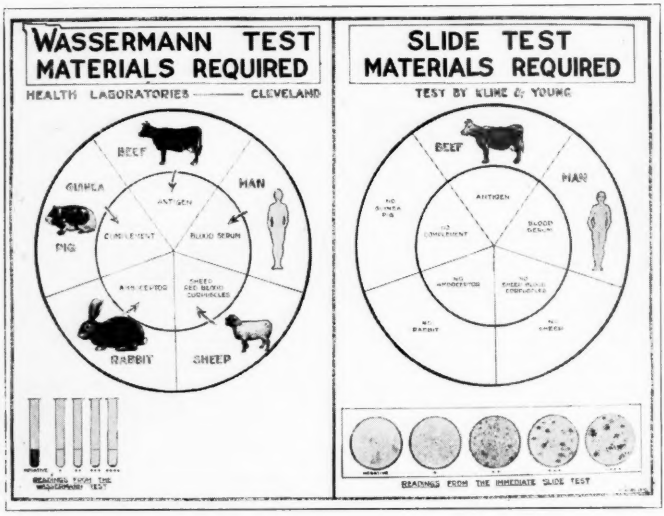
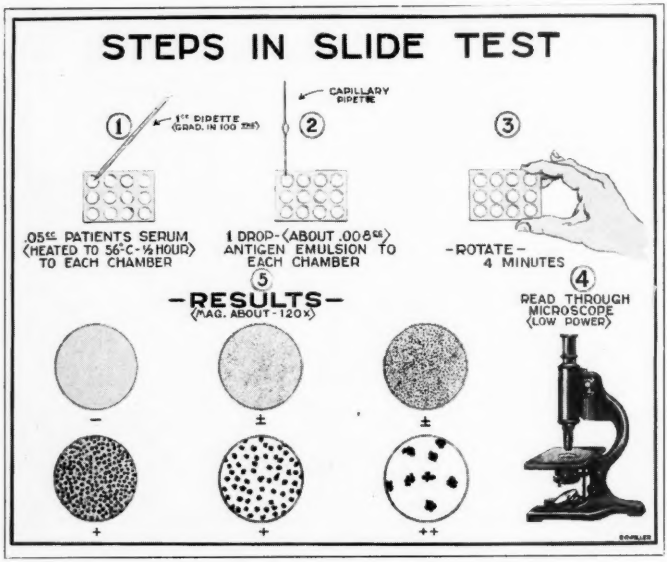


FIGURE 2


WASSERMANN TEST
MATERIALS REQUIRED
HEALTH LABORATORIES ——— CLEVELAND
BEEF
MAN
GUINEA PIG
RABBIT
SHEEP
ANTIGEN
COMPLEMENT
HUMAN SERUM
ANTIBODIES
SHEEP RED BLOOD CORPUSCLES
SLIDE TEST
MATERIALS REQUIRED
TEST BY KLINE & YOUNG
BEEF
MAN
ANTIGEN
BLOOD SERUM
READINGS FROM THE IMMEDIATE SLIDE TEST


STEPS IN SLIDE TEST
1
1ST PIPETTE (GRAD. IN 100 THS)
.05cc PATIENTS SERUM (HEATED TO 56°C - ½ HOUR) TO EACH CHAMBER
2
CAPILLARY PIPETTE
1 DROP - (ABOUT .008cc) ANTIGEN EMULSION TO EACH CHAMBER
3
- ROTATE - 4 MINUTES
4
READ THROUGH MICROSCOPE (LOW POWER)
5
- RESULTS -
(MAG. ABOUT - 120X)
-
±
±
+
+
++

A capillary glass pipette 5½ inches in length and 3 mm. in diameter is placed over the edge of the carrying case (see Fig. 1) at an angle of about 30°. The applicant's finger is then cleansed with alcohol, sponged dry and a puncture wound made with a sharp needle. The bleeding finger is squeezed and the blood is allowed to run into the capillary pipette until it is about three-fourths full. (This will yield about .2 cc. of serum which is sufficient for at least three Kline tests.) A heavy rubber band is now placed around both ends of the capillary pipette to prevent leakage and evaporation. It is replaced in the box and sent to the central laboratory for testing. In the laboratory the rubber band is removed and one end of the pipette is sealed with a Bunsen burner. A fine wire is then inserted into the open end of the tube to break up the clot.

The blood in the pipette is now centrifuged for thirty minutes at high speed (about 2,000 r.p.m.) which packs the clot into the lower half leaving the clear serum in the upper half. The pipette is then placed in a bath filled with water at a higher level than the top of the serum and kept thus at 56° for one-half hour. After this the pipette is filed and broken just above the clot. The serum is then allowed to run into, or is drawn into, a 1 cc. pipette graduated in .01 cc.

The tests requiring the least precautions are those with heated sera[2]. In these it is not necessary to determine the pH of the water and salt solutions, and furthermore the antigen emulsions are satisfactory for use for forty-eight hours.

Materials for the Kline Tests for Syphilis

Glassware: Microscopic slides 3 x 2 inches as purchased are rubbed on both sides with Bon Ami paste (prepared by breaking up a cake of Bon Ami in a small quantity of water). As soon as the paste is dry (in about five minutes) it is completely removed from the slide with a soft muslin cloth. Upon clean slides, 12 paraffin rings, each with an inside diameter of 14 mm. are mounted (see fig. 2). These rings are made with hot

parowax by means of a thread wound wire loop, 15 mm. inside diameter.

Pipets: The pipets needed for delivering the sera and those for preparing the antigen emulsions are the ordinary finely graduated 0.2 to 10 cc. pipets. The pipets for delivering the antigen emulsions are Wright pipets made from glass tubing 6 to 10 mm. in diameter with the tubes about 0.5 mm. in outside diameter, delivering a drop equal to about 0.008 cc. (62 drops per 0.5 cc.)

Slide Holders: The slide holder is a wooden lid of a slide box (3½ x 6¾ inches) containing an easily fitting thin wooden shelf having a small handle at each end.

Kline Antigen:* The antigen is a lipid obtained by precipitation in acetone at 50°C. to 37°C. of concentrated absolute alcohol extract of beef heart muscle powder (Difco). The details of its preparation are given in a previous report[1].

Antigen Emulsion: The diagnostic antigen emulsion has the following formula:

0.85 cc. distilled water.
1.0 cc. one per cent. cholesterin (C. P. Pfanstiehl in absolute ethyl alcohol, 99 plus per cent. The cholesterin solution is prepared in about 45 minutes by placing in an oven at 50° to 56° and shaking gently a few minutes at 15 minute intervals. Kept at 37°C. this solution is satisfactory for use for a few months.)
0.1 cc. antigen.
2.45 cc. 0.85 per cent. sodium chloride.

The emulsion is prepared as follows:

Into a one ounce bottle 0.85 cc. of distilled water is pipetted. The bottle is held at an angle and the 1 per cent. cholesterin in absolute ethyl alcohol (99 plus per cent.) is allowed to run along the side of the neck of the bottle.

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*Standard Kline antigen may be obtained from the La Motte Chemical Products Company, McCormick Bldg., Baltimore, Md.

The bottle is gently rotated from the neck for twenty seconds. The bottle is held at an angle again, and 0.1 cc. of antigen is pipetted against the side of the neck from a finely graduated pipette. The bottle is promptly stoppered with a cork and shaked vigorously (the fluid thrown from bottom to cork and back) for one minute.

The 0.85 per cent sodium chloride solution is then allowed to run in quite rapidly, the bottle is again stoppered and shaken fairly vigorously for one minute.

Place the antigen in a test tube in a water-bath at 35°C. for fifteen minutes. The emulsion as soon as heated is ready for use.

The emulsion when examined thru the microscope, at a magnification of about 120 times, shows numerous very fine particles but no clumps whatever.

Technic

1. Place three heated serum slides each with twelve chambers, on a tray in a small holder.

2. Into each of the 36 rings, pipet 0.05 cc. of the heated serum to be tested.

3. After all the sera are pipetted, 1 drop of the diagnostic test antigen emulsion (about 0.008 cc.) is allowed to fall from a Wright pipette into each chamber with the serum.

4. The slides in the holder are rotated on a flat surface for four minutes.

5. The results are read at once through the microscope at a magnification of about 120 (low power 16 mm. objective, eyepiece 12) with the light cut down as for the study of urinary sediments and reported in terms of pulses according to the degree of clumping and the size of the clumps.

Any spilling from the chamber makes the reaction therein unsatisfactory and when this occurs the serum should be retested.

Results

With the kind cooperation of the Metropolitan Life Insurance Company a series of tests were carried out on 110 individuals. The majority of blood specimens were obtained from employees of the various departments of the Company, but a few specimens were from applicants for insurance who came to the laboratory for examinations. The blood was obtained in all cases from finger puncture. At the same time, a sufficient quantity of blood was obtained from the arm in order that comparative tests might be done with the Wasserman and Kahn procedures. The individuals were divided into syphilitic and non-syphilitic groups. Eighteen (16.3%) of the blood specimens were obtained from individuals who were definitely syphilitic.

The evaluation of results in this comparative study was made according to the method of Kahn as follows: Agreement, positive or negative by both methods; relative agreement, positive or negative by one method and doubtful by the other; disagreement, positive by one method and negative with the other.

Comparison of 110 Diagnostic Finger Blood Tests with the Wasserman Test

(See Table 1)

In 92 non-syphilitic cases there were no disagreements between the results obtained by the finger blood and Wasserman tests; there was total agreement therefore in 100 per cent. In the 18 syphilitic cases there were two disagreements. Both of the disagreements were positive with the finger blood test and negative with the Wasserman reaction. We must remember that these were on syphilitic patients. Thus if the Wasserman reaction alone was depended upon, these two cases would have been missed. This demonstrates the specificity of the finger blood test, as well as its greater sensitiveness.

TABLE I

Comparison of

110 Diagnostic Finger Blood Tests and Wasserman Reactions

	Syphilitic Group		Non-Syphilitic Group	Combined Groups
			Agreement	Agreement
Tests	13		87	100
Per Cent	72.2		94.5	90.9
	Relative Agreement		Relative Agreement	Relative Agreement
Tests	3		5	8
Per Cent	16.6		5.4	7.2
		Disagreements	Disagreement	Disagreement
	Positive Finger Blood Negative Wasserman			
Tests	2		0	2
Per Cent	11.1		0	1.8
	Positive Wasserman Negative Finger Blood			
Tests	0		0	0
Per Cent	0		0	0
Tests	16		92	108
Per Cent	88.8		100.0	98.1
	2			2
	11.1			1.8
				Total Tests 110

TABLE II

Comparison of
110 Diagnostic Finger Blood Tests and Kahn Tests

	Syphilitic Group				Non-Syphilitic Group			Combined Groups			
			Disagreements								
	Agreement	Relative Agreement	Positive Finger Blood Negative Kahn	Positive Kahn Negative Finger Blood	Agreement	Relative Agreement	Disagreement	Agreement	Relative Agreement	Disagreement	Total Tests
Tests	14	3	1	0	90	2	0	104	5	1	
Per Cent	77.7	16.6	5.5	0	97.8	2.1	0	94.5	4.5	.9	110
Tests	17.0		1		92		0	109			
Per Cent	94.4		5.5		100.0		0	99.0		19	110

Comparison of 110 Diagnostic Finger Blood Tests and Kahn Tests

(See Table 2)

There was an agreement of 100 per cent. in the non-syphilitic cases. There was one disagreement in the syphilitic group with a resulting total agreement of 98.1 per cent. The disagreement was positive with the finger blood test and negative with the Kahn test, so that if only the Kahn test had been done this case of syphilis would have been missed.

No false positives were obtained by any method used which accounts for the 100 per cent. agreement in non-syphilitic cases.

Comment

Hazen[8] estimates that 400,000 cases of fresh syphilis develop each year in the United States, but only about 42 per cent. undergo treatment during the first twelve months. The majority of the remaining cases in time become free of the cutaneous manifestations and constitute the so-called latent or concealed syphilis. The incidence of syphilis necessarily varies with the race and social strata of the population. In a recent report on the incidence of syphilis in private practice among the white race, Kiser and Bohner[9] found that in 2,872 consecutive examinations on private patients who came in their office for physical examinations, 105 (3.65 per cent.) had straight four plus reactions for syphilis with Kahn and Wasserman tests. None of these patients came primarily because of known syphilis, in fact none had primary lesions at the time of examinations and only two had secondary manifestations. Thirty (28.5 per cent.) of these syphilitic cases were classified as having latent or concealed syphilis. In these latent cases no positive stigmas of the disease could be demonstrated, except for the positive serological findings. In the colored race Granger[10] reports 16.5 per cent. positive serological findings among patients in private practice. It is well known that positive serum reactions are sometimes the only definite evidence of syphilitic infection and are not infre-

quently the sole means for detecting syphilis in its latent and concealed stages.

It has also been estimated[8] that 85 per cent. of infected individuals who receive no treatment and live for twenty years after their infection develop some sort of cardiovascular syphilis. Many of the cases go on for years without their syphilis being detected. A certain number are diagnosed by virtue of the fact that they develop one or another of the later manifestations of the disease. In view of the incidence of greater morbidity among untreated syphilitics, it is clear that a diagnosis and treatment of the disease is imperative in all cases. This is of especial importance to insurance companies and any test that lends itself to routine use must prove invaluable to the insurance company. The finger blood test as here described seems ideal for the purpose.

The taking of a venereal history is practically useless in insurance examinations. Many latent syphilitics never know that they had the infection, and the majority of those who know they have or have had the disease deny it for obvious reasons. Thus many of these syphilitics will pass the examination either because the infection is in the latent stage or because the examiner is not sufficiently trained to diagnose syphilis in its rarer and concealed forms. That this is not merely conjecture is proven by the following. While working with transfusion boards where routine blood tests for syphilis are made, a surprisingly large number of professional donors with concealed syphilis were found, who volunteered the information that they had recently passed an insurance examination. It is reasonable to expect a certain percentage of those seeking insurance are syphilitics in the latent form, which in many instances could only be detected by a blood examination. Whether an applicant suffering from syphilis should be entirely refused insurance or whether there should be an adjustment of the premium, depends upon many factors, including the stage and type of the infection, the amount of treatment the applicant had already received, etc.

Dr. Schamberg, professor of Dermatology and Syphilology at the University of Pennsylvania, recently suggested to one of the largest and strongest life insurance companies, that it require a blood test as a precedent to the issuance of policies. He explained that this would only lessen the cost of premiums to non-syphilitic policyholders; would serve not only as an important educational program; would not only gain valuable statistical data on the prevalence of syphilis, but would discover to tens of thousands of people the fact that they are suffering from syphilis at a period when its ultimate terrors could still be obviated. The reply to this communication was that, after consultation, it was not found to be feasible.

Conclusions

A Kline finger blood test for syphilis is described which is particularly useful for the detection of syphilis in applicants for insurance:

(1) The blood is easily obtainable from a finger puncture.

(2) Only a small amount of finger blood is required.

(3) No chemicals need be added to prevent clotting or bacterial contamination.

(4) The finger bloods can be sent with safety over long distances to a central laboratory for examination.

(5) The finger bloods are satisfactory for testing even ten days after collection.

(6) This test appears to be more sensitive than the Wasserman and Kahn tests.

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Dr. Chargin—It is perhaps well to introduce the few remarks that I shall make, by reminding you that long before the serologic tests appeared upon the scene it was possible to recognize syphilis clinically. The presence of an initial lesion, secondariers, or the late manifestations of tertiary or cerebrospinial syphilis, or in the presence of an adequate history, serological tests were, of course, unnecessary. But there still remained a very large group of individuals with latent or asymptomatic syphilis, in which a diagnosis was extremely difficult, and it was here that the serologic tests have become so important.

With the advent of the Wasserman test in 1906, based upon the Bordet-Gengou reaction, you will recall that it was at first thought to be an antigen-antibody reaction, and therefore a specific reaction. You will recall that the antigen employed at first was congenital syphilitic liver in which there were an enormous number of spirochetes and which gave rise to the error that the antigen need be specific. But it was soon ascertained that more accurate results and a larger percentage of positive reactions were obtained with antigens that did not contain spirochetes. As you know the antigens now employed are as a rule from animal hearts. The complexity of the technique of the Wasserman test, and the ingenuity of investigators had lead to an attempt of simplification of the serologic test employed in the diagnosis of syphilis. This gave rise to the so-called precipitation tests or better still agglutination tests of

which the earliest are the Sachs-Georgi test and Meinicke test abroad and the Kahn test and lastly the Kline test in this country. Taken by and large all the tests so far devised are fairly accurate. Some more than others. The Wasserman test is in agreement with the clinical findings in about 90% of cases. The Kahn test in a similar group of cases is 95% accurate, while the Kline test is 97% accurate. Yet this does not tell the entire story. In a percentage of cases the precipitation tests by virtue of their greater sensitivity are liable to give inaccurate results. At the onset actual experience according to numerous authors have shown these precipitation tests to give false reactions in from 4 to 10% of the cases. But with more recent technic in the hands of experienced workers, this error has been so much reduced that we may now state that the Kline test is as accurate and in many respects superior to other tests, especially in view of its simplicity considered from every standpoint. A test to be of service especially in your work must be easy in the collection of specimens, simple in technic and accurate. A routine serologic test is of importance in your work, for with the single physical examination, the necessarily cursary history and the uncertain venereal history, the examiner is apt to miss the presence of a syphilitic infection. The test as here described by Dr. Rein appears to fulfill all requirements for insurance work.

Mr. Blaivas—Dr. Rein has gone into the simplicity of this Kline Test, but as a laboratory man, we don't just look for simplicity. As you know, a very important factor is the accuracy of the test, which was brought out. The amount of blood is so small, that is, .05 cc. that we have no difficulty in obtaining that blood even from the finger.

One very important factor in the Kline Test that I found is the fact that the test is completed in a very short time. Dr. Rein mentioned transfusions, and I had a very interesting experience only a short time ago. We were piping some blood for a transfusion. The donors in New York City have a Wasserman done every thirty days. The donor presents a card with a

negative Wasserman and the active type and while cross matching, I decided to do a Kline Test and I find a 4 plus reaction. The man denies any luetic infection. I refused to let the transfusion go on. Later on the individual tells us that only a week after his blood Wasserman by the Health Department in New York, he had a primary lesion which we picked up, so that I merely demonstrate this or show this to demonstrate the importance of the Kline Test as far as the rapidity goes. We were through with our Kline Test about the same time we were through with the piping of the transfusion.

One last and very important factor that I want to bring to the mind of the insurance companies is the standardization of the technique, something that we have never been able to do serologically in our Wasserman work. The sheep blood may vary, the amboceptor (?) may vary and when I do a Wasserman in New York and somebody else does it in Philadelphia with identically the same technique, our results may differ. That has been the great drawback in serology as long as I can remember. With the Kline Test, where the only important factor is the antigen and nothing else to consider, we have a possibility now of standardizing our technique so that the results will be the same throughout the country. I thank you.

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